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XXXVI

ON BEING A PROFESSOR EMERITUS

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Everything comes to an end except walking a dog. It has long been the custom in universities, when a professor reaches the age limit, to retire him with the title of Professor Emeritus. It is the kiss of death. Conferring the title is the academic form of execution. You and the other victims of time are given a place of honor on the commencement platform much like that of a corpse at a funeral. You rise when the President of the University comes to your name on his list and bow your white head, to all outward appearances submissively. The title conferred, you are then legally and academically dead.

To step out of the ranks, to see your place quickly, almost automatically, filled; to hear the sturdy tread of your companions of yesterday fade away in the distance, this—the books say—is life. I now agree. Fortunate is the man whose accumulated philosophy fully covers the occasion.

When your Chairman asked me to be one of the speakers this evening, I chose as the subject of my short talk "On Being a Professor Emeritus." Perhaps a better title would be "Reflections of a Professor Emeritus." What I have to say consists of a number of

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observations concerning the state of our specialty, certain comments on the running of a Department of Otolaryngology, the airing of a few miscellaneous opinions of mine on topics concerning the specialty, ending with some ideas borrowed or rather quoted from Professor Perry's pamphlet "A Plea For An Age Movement." Finally I offer a set of rules of conduct for a retired chief-of-service. Much of what I shall say will sound like preaching. Frankly, I intended that it should.

Let me begin with a rather childish confession concerning this retirement business. I felt acutely that the title of chief was no longer mine. It had naturally and legitimately passed to another. I got more silent satisfaction out of this title than that of professor. There was a certain friendliness about it and it was spoken now and then, I imagined, tinged with a touch of affection.

At the Harvard Medical School and at the hospital we seldom use the title of professor. You are simply Doctor to your fellows and to your students. I don't know what the custom is in other parts of the country or at Yale and Johns Hopkins. What I remember most about Johns Hopkins is that Osler almost made it human, and I like to recall a fact about Yale which you seldom hear, namely, it was founded by two graduates of Harvard.

So much by way of introduction. Now for more serious things.

Improvements. Be prepared to see improvements, perhaps as soon as the first week after you retire. You will be startled to find that some of them were crying to be made and your successor quickly saw the need of them, and you didn't. This happened to me. Take the improvements with good grace and praise them.

The Specialty at the Crossroads. The world has narrowed, thanks to the wireless and the airplane. The surgery of our specialty, and our specialty has been proudly surgical, is shrinking due to chemotherapy, and the end is not yet in sight. The specialty is at or near the crossroads. My advice to a young man training for the specialty is to equip himself to do neck surgery or do what men of two generations back did, take up in connection with the specialty diseases of the chest or even chest surgery. Bronchoscopy will soon be swallowed up by thoracic surgery. Today to most men laryngology alone is too narrow a specialty unless combined with otology. Otology is always on the edge of brain surgery. Therefore, the modern otologist should have a working knowledge of neurology and be proficient to a certain extent in brain surgery. In

my opinion, he should be competent to open a cerebellar abscess either by the internal route, that is, from the mastoid, or by the external route, through the occipital bone. At present he is required to do only half of the job.

Training the Staff. Give the men under you increasing and progressive opportunities to operate. Then when you retire you leave a staff trained to do everything which you can do, even some things which you have not done. This happened to me. Men grow with responsibility and are measured by it. Don't be a hog. This has happened and is happening. If you become jealous of the younger men don't show it. Or better, conquer human nature and don't let it get a hold on you.

At times your endeavor to give each man a progressive chance is not entirely good for the patient. Men learn not by doing once but by repetition. First operations, however, are not always full successes. Assigning certain operations, say the radical frontal, or cases of brain abscess to one man exclusively for a year or more should eliminate partially successful first operations. This may upset your staff, but the good of the patient should outweigh personal ambitions. Giving a progressive opportunity to operate within the limits I have mentioned is a great builder of esprit de corps, or to use a word more popular today, of morale.

Another morale builder is to bend over backwards to give due credit for ideas or help of any kind. When the mind has fondled an idea even for a short time, it often seems like its own child. Even a house officer may be the father of a pregnant thought. If you credit him with it, he will forgive almost anything you may feel called upon to do to him. If you forget, it will rankle in his heart for life, and automatically you lose his full respect. This bit of preaching applies equally strongly to your dealings with all members of your staff, and to the staff individually.

Teaching. All teachers should be paid. Theoretically the best teacher should be the full-time man. In teaching of any type nothing is more disastrous than to have the instructor come in breathless from his office and have him look at his watch from time to time in order to see when he can hurry out. This is especially true in postgraduate instruction. The graduate is giving his whole time to the course and besides, his tuition, living expenses and loss of practice represent a very sizable sum of money. I early gave up my private practice while giving my postgraduate course.

Friday Nights. I have spent my whole medical life surrounded by young men. As I look back upon what I have tried to do for House Officers, the thing which has paid the greatest dividends was the Friday Night Exercises at the medical school. I kept them up for at least thirty years, the only break occurring during the last war. The part which the House Officers played in the meetings was to abstract current medical literature. I picked out the articles. The House Officer is charge of the evening assigned them. All of the men took it seriously. In the beginning some were nearly made sick by it. Often their first attempts were painful for them, and for me. I did not pose as a perfect example. They could easily see that I was not. The point was not what could I do but what could they grow to do. I praised them when I could. This was often with those who had been at it for a while. They were criticized freely. If they were bumptious and feebly facetious, perhaps they were criticised severely. I criticised everything about them, including their voice, the way they stood, and their mannerisms. I reminded them that these Friday Nights were the only place where they would get criticism to their face and not behind their back. At the end of their time with us I honestly envied many of them. Some of you have heard some of them talk. You will hear one tonight. You may think I was trying to act as the Lord's first assistant. I was. At times he needs one. More than one house officer has told me that nothing which happened to him while he was with us helped him more than those exercises in speaking on his feet.

By carrying out this program the house officers did my reading for me. Now I do it for myself and am always behind in it. I have not yet mustered courage to cut down the number of medical journals I take. I take too many and they come too often. Medical journals which come weekly at times are almost a pest. The paper shortage as yet has had little or no effect upon them.

Speaking of medical journals, I might add that in many of ours the advertisements shriek of high pressure salesmanship; a full-page advertisement is hard for the editor to resist even if it skirts the edge of the ethical, and the paper of some journals blazes like a celluloid collar. The illustrations are usually splendid, but the polish put on the instruments and rubber gloves is not always equalled in the reading matter. To me it is a delight to turn to the Lancet or the British Medical Journal. In the latter you are even tempted to browse about. Few if any of our journals invite this.

The Basic Sciences. Today you hear much about the basic sciences and the specialties, especially from the Advisory Board of

Medical Specialties. It is easy to become a snob or a Holy Joe in this matter. The specialty which requires the fullest training in the basic sciences at present is surgery. Unfortunately our specialty does not rate very high. It is up to us to give better instruction in the basic sciences. The responsibility of doing this is squarely on the shoulders of the full-time chief of service. More than one of the better known hospitals have staffs who can give this instruction without calling upon the help of the associated medical school. Whether you consider it bragging or not I will say that the Massachusetts Eye and Ear Infirmary can do it in the specialty of otolaryngology.

The Fenestration Operation. I am waiting for the five and ten year results of this operation. It is clear that it is not an operation for the occasional operator. My prediction is that 15 to 20 per cent of these operations will be permanently successful. They are all immediately and temporarily so. This is old knowledge, and this is the possible tragedy of the operation. To regain one's hearing so dramatically and then to lose it is literally to drop from heaven to hell. For a man who has real worthwhile work to do in the world, to regain usable hearing for three to five years is more than worth while and justifies the operation. It looks at times as if we should have to recast our knowledge of the physiology of the ear on account of this operation.

I have often wondered this: suppose Beethoven had had otosclerosis instead of a toxic nerve deafness and had a fenestration operation with a temporary or a permanent result, how the world of music would have been changed!

The Radio. I have always thought that the three chief occupations of man were to eat, sleep and beget. I would now add a fourth, namely, listening to the radio. If a statement is repeated often enough, nine out of ten people will believe it. We are born believers. This is the chief trait of the mind of the savage. With us it is an infantile trait which we have not outgrown. Until the Greeks introduced doubt or skepticism, knowledge did not start on its upward path. Speaking of the radio, our old stand-bys Lydia Pinkham and Carter's Little Liver Pills have appeared on certain stations. Doctors and laymen now seem to get their therapeutics from the radio, not from the medical schools. I imagine that the time is far off when the besmirching of broadcasts by the advertising of drugs will be cut down or abolished because more than one medical school owes a large part of its foundation money to gifts or bequests harking back to patent medicines, or tobacco concerns.

The Laboratory. A blueprint of the arrangement of the rooms of the Department of Otolaryngology at the Massachusetts Eye and Ear Infirmary shows that the pathological laboratory is on the ground floor, and geographically in the center of the department. As a rule the laboratory is isolated in a far-off corner, often in a separate building. Fortune dictated the arrangement at the Infirmary when the Throat Department of the Massachusetts General Hospital joined with the Ear Department of the Infirmary. This central location of the laboratory to me is ideal, and worth copying. Not having any morning office hours I spent the morning at my desk in the laboratory. The X-ray Department and Dr. MacMillan were opposite and only a few steps away. From the laboratory I carried on the administration of the department. Here I received visitors. Members of the staff on duty and off were continually coming and going. So were the House Officers.

The room is large enough for the weekly clinical meetings of the staff. Visitors were welcome except at the monthly mortality meetings. These were for the staff alone because this meeting hews to the line letting the chips fall where they may.

Permit me a word about the house furnishings of the laboratory. There is the usual lantern for the projection of microscopic slides and another lantern which came later and is the joy of my heart. The coming of this doubled the interest of the clinical meetings. It projects lantern slides, x-ray films of sinuses and mastoids, drawings, book illustrations, and temperature charts. None of these have to be passed around. Dr. MacMillan demonstrates the x-ray films and everyone in the audience can see the enlarged picture of the film as it is shown on the prepared surface of the wall. We soon discarded our special screens. I cannot help adding at this point that the petrous tip which in the x-ray film was always darkest Africa to us, now often startles us by what a good plate will show as interpreted by Dr. MacMillan. The speaker's platform has a microphone and a loud speaker. This means that everyone in the back of the room can hear, and it helps the older members of the staff who are hard of hearing and who sit in the front row. There is a hand microphone which can be passed to anyone in the audience who wishes to ask questions. The above sounds like the "short and simple annals" of the kitchen, but the kitchen is the heart of the house three times a day, sometimes oftener, if there are children about. At times doctors act like children.

Sinus Operations. I should like to see a country-wide report on the epidemiology of sinus disease and osteomyelitis; also a collective

report on the results of radical sinus operations covering a period of at least ten years. There is no more pitiable patient than the over-operated sinus case. All of us have had them. Proetz has lately reminded us that we have often forgotten function. In our zeal to do what we proudly call a complete anatomical operation, provided that there ever is such a thing, at the risk of being called conservative, a slur which is handed round freely by the so called he-men of the specialty, I feel that we should stop for the pause that refreshes and listen a bit to Proetz. It is well to remember that conservatism is often another name for good judgment; it is not always timidity.

Chemotherapy. At the moment we are in a highly emotional mood in regard to chemotherapy. The same can be said of vitamins. The cod liver oil of our boyhood days, the yeast of our mother's bread, and even the mold which sometimes embarrassed her when it appeared, all these are revealing untold treasures to the biochemist. I envy those who will have the opportunity to see how far these miracle agents will carry and to evaluate their final and over-all accomplishments. Not for me, but for you, is the great adventure.

Shortly after flying his kite, Franklin was asked to outline his view of the future of electricity. He replied that "he was born too soon." Some of us would have to make the same reply if asked to predict the value of the swarm of advances now being made in medicine. Also, we would have to make the same reply if asked about social medicine. Russia, you know, has already gone to the limit in this matter. A detail which you may not know is that it offers physicians refresher courses every three years at state expense. Should such a thing happen here, some of us who give graduate courses would suffer both in fame and fortune.

While on the subject of Russia it is well to remember that in 1892 a young man named Iwanowski read a four page paper, "On the Mosaic Disease of the Tobacco Plant," in which in two sentences at the end he recorded the basic observation which six years later led to the discovery and study of virus diseases. Virus disease was held to be nonbacterial in origin, but due to an active agent which was "a contagious living fluid" which passed every known filter. In medicine, therefore, Russia has much to its credit. Those who have seen its present day war surgery say that it is superb.

Please pardon a few sentences in a rather nostalgic vein. While actively connected with the hospital I felt a sense of security about my family and myself in case of accident or illness. My medical friends were in the same group with whom I had grown up

or had watched grow up. I knew their abilities and they knew my idiosyncracies. We, as it were, belonged to the same lodge. Now I am twenty miles away, which is a long ambulance ride. I know intimately none of the physicians in the town where I now live. It is often a severe emotional upset for a patient to lose the friendship and the guidance of a physician whom he has long trusted. There is a strong sense of loneliness about it. At my time of life my greatest desire is to see the ones who have travelled life with me through to the end in familiar surroundings and with familiar faces about. Such thoughts as these bring to my mind our rural communities—and there are many of them—which because of the war either have no physician, or have lost their regular physician. The people of such communities must feel more than forgotten.

Now that the end of what I have to say is in sight I should like to refer again to the title of Emeritus and its connotation of age. Shortly before Christmas, Dr. Hill, on whose convenient shoulder I have at times wept, sent me a little book called "A Plea for an Age Movement." It is written by Professor Perry of the Harvard Department of Philosophy. I would almost give my soul to be able to think and write as he does in this pamphlet. It is wise—one has to be wise to be a philosopher—and deliciously witty. Dr. Hill said that it might be a comfort to me, and it has been. Dr. Lierle, to whom I sent a copy as a Christmas remembrance, after reading what it said about the young, replied that it was so true that it ceased to be funny. I urge you who have children to read the book. Those of you whose hair is getting white should find it very much worth while. Dr. Perry's main idea is that age should not allow itself to be shelved by youth, but keep in the game and still do enough to be counted.

Let me quote a little from Professor Perry. This done I will get back to my main subject and close by giving a set of "Rules of Conduct for a Retired Chief of Service."

"We have heard a good deal recently about the 'youth movement' and I suggest that the time has come to start an 'age movement.' There was a time when old men held a good position in the world.

"The theory was that although we had slowed down physically, and although our arteries had hardened a bit, and although our wind was short except for talking, and although we had lost something of our sex appeal, we had more than made up for it. We were supposed to have laid by stores of wisdom, so that we could offer good counsel.

"We were supposed thus to be qualified to be the rulers of our wives, the mentors of our children, and the elder statesmen of the realm.

"Recently we have fallen to an all-time low. We are retired at an early age from business and the professions. We are hustled by our juniors in politics. And

as to the armed services, it is universally held that what they need is young officers. The Joffres and Hindenburgs are regarded as accidents. The success of the Germans is attributed to the youth of their officers, the failure of the French to the age of theirs. What is needed, we hear on every side, is young blood."

"The most striking evidence of the downfall of the aged is to be found in the domestic circle. The authority of the father was first broken by the mother, and the children poured through the breach. The last remnant of paternal authority was the period in which the father was an ogre, who came home at the end of the day to deal with major offenses and who could be invoked by the mother as a threat during his absence. Although he was no longer magistrate, he was at least executioner. But even this role disappeared when domestic criminology was modernized, and the child's insubordination was regarded as a personality-problem to be solved by love, hygiene, and psychoanalysis. The father, knowing neither physiology nor Freud, and having been denied all natural affection in order to serve as the big stick, now played no part whatever in the civil order of the home. As head of the family he went definitely out, along with such ideas as naughtiness, punishment, discipline, and obedience. He remained, of course, as bread winner, chore-man, and stud-horse, but these functions carried no prestige. The mother, who had conspired with the children to break down the authority of the father, lived to regret it. She suffered from the same age disability as the father and she was more continuously exposed to its consequences. The general outcome was that both parents found themselves on the defensive."

"Those who are old enough to remember several wars are supposed on that account to be disqualified from judgment about this one. Those whose judgment is respected, those who are supposed to know what war is, are those who have never experienced war and who have even forgotten their history. They tell us, for example, that wars never settle anything, and they are supposed to know. The elders, being rejected from military service for physical reasons, may not offer counsel lest they be suspected of a sadistic desire to sacrifice the young."

It is hard to stop these quotations because there are many more meaty passages which apply to the subject in hand. I hope I have read enough to send you to the book itself.

RULES OF CONDUCT FOR A RETIRED CHIEF OF SERVICE

(Not taken from Chesterfield)

1. Keep away from the hospital for six months or a year until the new man is comfortably settled in his job.

2. Expect improvements. Praise them when they come.

3. When your successor is presiding at a meeting, don't sit under his nose.

4. Never look surprised.

5. Don't snoop.

6. Give advice only when asked. Don't expect to be asked.

7. Make no comparisons; listen to none. They may be uncomplimentary to you.

8. Don't feel openly hurt if your pet instruments, especially those you invented, are not used, but are put in the historical collection. Don't embarass your hospital by leaving behind or by giving it anything too personal. Sentiment hampers administration.

9. Don't ask if you are missed. Out of politeness the answer will always be "yes" whether true or not. Should any one voluntarily say that you are missed, it will be sweet music to your ears for that is what you long to hear.

Of course I have broken some of these rules. It was only human to do so, and I have been accused of being more human of late. However, each slip that I was conscious of brought appropriate chagrin.

I have given these rules because they soon may be of use to some of you. Many of them apply to the active Chiefs-of-Staff. That is, they are retroactive.

As my closing sentence allow me to sum up my experience as a specialist as follows: The strength of specialism is specialism. Paradoxically its weakness also is specialism.

127 FRONT STREET.

XXXVII

PATHOLOGY OF NASAL MUCOUS MEMBRANE AND SUGGESTIONS AS TO TREATMENT

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From the title, this paper falls naturally into sections; the first on the tissue changes of nasal mucosa is somewhat scientific; the second, dealing with treatment, is, I hope, practical.

Respiratory mucous membrane consists of epithelium, basement membrane, and a tunica propria or stroma.

The epithelium is of a pseudocolumnar ciliated variety, and in the embryo and infant is uniform throughout the respiratory portion of the nasal mucosa. In the adult, from repeated nasal infections, from traumatization of respiratory blasts of air or from trauma of faulty nasal hygiene, nature may try to protect traumatized areas by a metaplasia from columnar epithelium to stratified squamous epithelium. Because of the absence of cilia, these are the so-called silent areas of the nose and are found over the anterior tip of the middle turbinate, the anterior tip of the inferior turbinate, and the inferior portion of the septum. The natural motion of the cilia is toward the ostia, and in the normal nose the surface epithelium is covered by a thin sheet of mucus.

The surface cells rest on the basement membrane. This thin band of undifferentiated tissue limits the epithelial cells from the underlying tissue. Its exact origin is doubtful but it is thought to be derived from connective tissue. It is canaliculated, and through the canaliculi, wandering cells may escape to the surface.

The tunica propria consists of a fibro-elastic connective tissue network—superficial, loosely interwoven, deep, more compact and condensed. Where the mucous membrane overlies cartilage, the condensed deep layer of the stroma forms the so-called perichondrium and, where it overlies bone, the periosteum. The tunica propria is

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the supporting structure. It gives support to the glands, vessels, nerves and lymphatics. Within the stroma are various tissue cells—polymorphonuclear leucocytes, histiocytes, endothelial cells, fibroblasts, connective tissue cells, and the small round lymphoid cells. The specialized venous cavernous spaces are particularly numerous in the inferior turbinate, to a lesser degree in the middle turbinate, and may also be found at the tubercle of the septum.

In acute rhinitis the normal histology is disturbed. The temporary vasoconstriction of the vessels is followed by a vasodilation. The polymorphonuclear leucocytes at first lag along the vessel wall, then cling to the wall and many by diapedesis pass through the wall and appear in the tissue spaces. The defense mechanism of the tissue becomes active, the wandering tissue cells migrate to the source of irritation, so that there is an increase of the wandering cells in the mucosa. With the slowing of the blood stream the blood plasma by transudation escapes into the tissue spaces. The cavernous spaces are dilated and temporarily lose their power to contract. With an excessive blood supply to the glands there is an excessive stimulation of the glands with a consequent excessive amount of secretion.

No two rhinologists agree on the treatment of an acute rhinitis. Although they all agree that the ideal treatment is bed rest at the first sign of a cold, the local treatment varies from those who perhaps overtreat the local condition to those who condemn all local medication. I am opposed to permitting or encouraging a patient to make a trip to the doctor's office on a cold wintry day to receive a treatment for a head cold. In my opinion, the first principle of treatment is nasal ventilation.

The late Dr. D. Campbell Smyth taught that vasoconstrictors should be used often enough to open the nose and then used often enough to keep it open. This is, I believe, good therapeutics. The vasoconstrictors should be isotonic and of a pH compatible with that of the nasal secretion. They should not irritate. The radio announcement that the stinging of the nose drops means they are penetrating the tissues is a gross misstatement of fact. Nasal vasoconstrictors should not sting or irritate. Test your medication on yourself and find out how often you could not or would not take your own medicine.

Vasoconstrictors contract the dilated vessels and cavernous spaces. The blood supply is diminished and shrinkage of the swollen mucosa permits nasal ventilation. With the diminution of the blood supply there is less stimulation of the glands and therefore less secre-

tion. Man, like the horse that can't breathe through its mouth, is more comfortable if he can breathe through his nose. George Catlin, the artist, after his life with the Indians, wrote in 1861 a small book entitled "Shut Your Mouth." You can only shut your mouth when you can breathe through the nose, and medication that permits nasal breathing is, in my opinion, medication based on nasal pathology.

I have not been impressed with the efficiency of bacteriostatic or bacteriocidal drugs used in conjunction or in combination with vasoconstrictors. Limited bacteriological investigations of their efficiency in a selected group of patients has failed to show any demonstrable diminution of bacteria by their use. I am not sure about the efficiency of the use of finely divided sulfonamides insufflated over the nasal mucosa after careful shrinkage. Some patients appear to be benefited by their use.

When the nasal secretion is so thick and sticky that the patient cannot properly clear his nose, I am not opposed to a gentle irrigation with Ringer's solution, but I am opposed to the use of nasal irrigations by the patient. I believe their use starts a vicious cycle and that they do more harm than good.

Spot suction is of great value in freeing the nasal cavity of excessive secretion. Massive suction is dangerous, as it may cause further trauma to a diseased membrane.

The correction of a coexisting constipation and the use of a "Dover's Powder" are two old remedies that have proven their worth.

Chronic Rhinitis—In chronic turgescent rhinitis the pathology of the nasal mucosa is characterized by dilated cavernous sinuses that have lost their power to contract. Associated with it there is an increase of the fibroconnective tissue of the stroma. The polymorphonuclear leucocyte has disappeared from the tissue spaces and there is an increase of the small lymphocytes, particularly around the glands and the ducts of the glands.

The increased vascularity of the mucosa means increased activity of the glands. The secretion is tenacious and sticky and with a blocked nose there is postnasal dropping and increased postnasal secretion.

The treatment depends upon the etiology, and the etiological factors are many. Jarvis' classification of causes is practical and should be utilized. A nasal airway unilaterally obstructed by deviations and spurs of the nasal septum will in time by overwork of the

open side cause a compensatory hypertrophy of the mucosa of that side. It is the blocking of the former good side of the nose that brings the patient to the physician. Climatic changes in temperature and in humidity; congestion of other body organs—such as the congestion of the stomach mucosa from overeating or overdrinking; congestion of the uterine mucosa from menstruation or from pregnancy; congestion due to constipation or congestion due to a tired out nasal mucosa from trying to fulfill its physiological function, not only all day but all night long—from the mistaken idea that to be rugged and robust you should let the wintry blasts blow over you —all these affect the nasal mucosa. We too often forget that of all the animals man is the only one that does not protect his nose while he sleeps.

The treatment of chronic turgescence of the nasal mucosa should be based on the correction of the etiological factor or factors producing the congestion. Normal nasal functioning should be restored by equalizing nasal ventilation by the correction of septal deviations and spurs, by correcting dietetic errors and by eliminating constipation. We should emphasize the relation of skin temperature to nasal congestion although we will not succeed in persuading anyone to dress properly. Bare legs, bare heads and bare chests are in fashion, even if the nose runs and the secretion trickles down the throat.

As many of my patients have been relieved by the simple expedient of partly closing the windows and sleeping in a room that is not under 50° F., as by any other means.

When anatomic, physiologic and dietetic errors are corrected, improvement ofttimes is hastened by destruction of the dilated cavernous spaces. This may be accomplished by subepithelial electrocoagulation or by the careful injection of sclerosing fluids into the stroma.

Vasomotor Rhinitis—The pathologic changes produced by vasomotor rhinitis are expressed by a swelling of the superficial epithelial cells, at times by a desquamation of the cells, by an alteration of the basement membrane, and the condition is characterized by dilated tissue spaces caused by transudation of serum into the stroma. There is an infiltration of cells, not only eosinophiles but also small round lymphocytes, histiocytes and plasma cells, an infiltration most marked just beneath the basement membrane. The cavernous spaces as well as the capillaries are dilated.

To enter into a discussion of the treatment of vasomotor rhinitis is beyond the scope of this paper. Suffice it to say that after a careful search for a specific allergen has failed, or when after an exhaustive effort to produce immunity by adequate treatment of suspected allergens, then certain local measures are of value. Vasoconstrictors give but temporary relief. Realizing that the basic pathology is one of edema with dilated cavernous spaces, subepithelial electrocoagulation or destruction of the dilated spaces and cavernous sinuses by the careful injection of sclerosing fluid seems logical. Small reactionary polypi may likewise be destroyed by the careful use of the electrocoagulating needle.

Atrophic Rhinitis—The etiological factors of atrophic rhinitis will not be discussed. The pathology is that of a metaplasia of the pseudostratified columnar ciliated epithelium to that of stratified squamous epithelium. The basement membrane is thickened. The stroma shows an increased fibrosis. The fibroconnective tissue becomes increased, the vessels are thickened and the glands, because of both the decreased blood supply and the fibrosis, undergo atrophy. Because of the absence of cilia the secretion stagnates, secondary infection occurs, and scabs and crusts form. With the complete atrophy of the glands and when there is no longer any glandular secretion, the scabs, crusts and odor disappear, so that atrophic rhinitis is said to be a self-limited disease.

Many therapeutic agents have been tried in this disease—at one time glucose applied locally on strips of gauze, at another the local use of estrogenic substances. At the present time, the local use of prostigmin is being used. Glucose and estrogen appeared to give some relief while the patient was under active treatment, but the scabs and odor re-appeared as soon as the frequent cleansing of the nasal cavities ceased. At the Infirmary we are attempting to duplicate the results of Soskin and Bernheimer[1] and substantiated by Henner and Busby[2] by the use of prostigmin methylsulfate.

I am of the opinion, however, that careful cleansing of the nose is the best therapeutic measure we have, although I admit it is treating the effect and not the cause of the disease.

Operative procedures to narrow the wide nares have in my hands failed.

SUMMARY

The histology of respiratory nasal mucosa, the pathological changes seen in acute rhinitis, chronic turgescent rhinitis, vasomotor

rhinitis and atrophic rhinitis, together with the highly controversial subject as to their treatment, is herewith presented.

243 CHARLES STREET.

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XXXVIII

INTRINSIC ALLERGY AS IT AFFECTS THE EAR, NOSE AND THROAT: THE INTRINSIC ALLERGY SYNDROME

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In any paper on allergy nowadays an explanation of what is to be included under the term should be attempted. Therefore I shall comment first on intrinsic or physical allergy. With this I shall mention briefly certain background subjects, such as desensitization, the alarm reaction and the known functions of the adrenal cortex, before I describe the syndrome which I have chosen to call the "syndrome of intrinsic allergy."

The first recognized allergic reaction, the tuberculin reaction, was of the antigen antibody type, and as pointed out by Kahn,[1] was a cellular rather than a humoral reaction. Kahn expressed the belief that allergic reactions of tissue are hyperimmune reactions in which the combination of antigen with antibody not yet released from the cell produces cellular disruption. This describes the so-called immunologic or antigen antibody type of allergy.

A great deal of work recently summarized by Rackemann,[2] Kellaway[3] and Farmer[4] suggests that many, if not all, of the phenomena which are recognized as allergic are produced by cellular injury with release of histamine which has been shown to be a normal constituent of most tissues. Histamine released into the tissue spaces acts on the arterioles and capillaries and produces arteriolar constriction and capillary dilatation in human beings according to Wagener[5] and also in animals according to Dale and Laidlaw[6] and Best and McHenry.[7] In addition, the permeability of the capillary walls increases and localized edema occurs subsequently.

Lewis and his associate[8] showed that in the skin of human beings sensitized by exposure to physical irritants, such as ultraviolet light and burning, further exposure to such irritants after a latent period

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will produce a reaction indistinguishable from that produced by the injection of histamine. Kellgren[9] felt that the production of a painful region in muscles, known as myalgia, by exposure to physical stimuli was a phenomenon similar to the cutaneous reaction produced by physical agents.

Lewis, Grant and Marvin[10] showed that, following stimulation of the skin of patients sensitized by physical agents, a condition of partial or complete refractoriness to further stimulation of the same kind was established. As this condition of refractoriness became established, the increased permeability of the capillaries of the skin disappeared.

Rose[11] stated that it was possible to distinguish between the immunologic or antigen antibody type of allergy, examples of which are asthma, hay fever and other conditions based on immunologic reactions, and a second type of allergy, namely physical or intrinsic allergy which is not believed to depend on an immunologic reaction. Sensitivity to heat, cold[12] and changes in atmospheric pressure, endocrine dysfunctions[13, 14] and emotional disturbances[15-17] are examples of intrinsic or physical allergy. Code[18, 19] has shown that in experimental anaphylaxis in animals histamine is liberated and Rose pointed out that the evidence on release of histamine in association with allergic phenomena, indirect though it is, has been found in patients who have had the second type of allergy. Evidence of a more direct naure has been obtained by Rose also. With the method devised by Code the histamine content of the blood was measured in a group of normal persons and was found to remain fairly constant. In a group of cases of intrinsic allergy Rose found the histamine content of the circulating blood may be increased after stimulation of the skin by exposure to the appropriate stimulus.

Hanhart[20] found in investigations of 70 large families in which there was a history of allergy that the general idiosyncratic disposition toward intrinsic allergy was inherited as a Mendelian dominant. He stated that although severe hay fever with pollen asthma may occur in well-balanced persons, perennial vasomotor rhinitis with bronchial asthma (a representative of the intrinsic allergy group) nearly always is indicative of a neuropathic or psychopathic personality and an unstable autonomic nervous system. He concluded that the hereditary disposition is far more important in the occurrence of the physical or intrinsic group of allergies than external influences, such as local sensitization through inflammation or trauma.

Berger[20] stated that a definite group of nonallergic causes consisting of physical, chemical and hormonal conditions and psycho-

genic reflexes is associated with allergic conditions. Any of these may produce any of the allergic conditions. The characteristic feature in such conditions is a commonly inherited or acquired constitution of certain tissues, particularly of the peripheral blood vessels, the connective tissues and the nerve ends. These tissues have a definite susceptibility for selected and preferred stimuli and tend to respond in typical ways.

Muzio[21] has shown that the immediate response of nasal mucosa to changes in temperature affecting the skin is mediated through the autonomic nervous system. Larsell and Fenton[22] have submitted evidence to suggest that intrinsic allergy, particularly as it affects the nose, produces the same effect as that which would be obtained by stimulating the vidian nerve after removal of the superior cervical ganglion and after sufficient time has elapsed for degeneration of the fibers from the cells in the superior cervical ganglion. They felt that the clinical picture of congestion of the nasal membranes, with watery discharge, associated with various disease conditions may have its physiologic explanation in the overflow of vasodilator impulses to the nasal mucous membranes. They[23] suggested that when such overflow is accompanied by abundant nasal secretion, the overflow is produced by the parasympathetic or cranial autonomic fibers since the secretomotor fibers belong to this system.

Grant, Pearson and Comeau[24] showed that urticaria could be provoked in certain persons by both psychogenic and physical influences. These authors concluded that H substance (histamine) was released from cells of the skin through the action of peripheral nerves belonging to the parasympathetic or cholinergic nerves. Grant and his co-workers felt that the release of acetylcholine in turn led to the liberation of histamine from the skin cells. They did not consider that their work indicated that the activity of the nerves involved was in any way abnormal. They felt rather that the skin of their patients responded abnormally to a normal release of acetylcholine at the nerve endings. They also found that a portion of skin of sensitized persons in which a wheal had been raised by the action of nerves failed again to respond with a wheal until after an interval of about two days. They felt that they might say that the urticaria is due to the skin cells having acquired a sensitivity to acetylcholine and that, when the cells once react with it, this sensitivity is discharged and requires a period of about two days for its re-establishment. They furnished evidence that the unresponsiveness was to some extent specific for the stimulus provoking it.

This evidence in favor of the cholinergic origin of urticaria in sensitized persons may be compared with that submitted by Larsell and Fenton in regard to vasomotor rhinitis. It seems a reasonable assumption that if a similar mechanism can be found as the basis of two manifestations of physical allergy, that all manifestations of physical allergy are probably mediated by a similar mechanism, that is, the setting free of histamine from certain abnormal cells by the action of cholinergic nerves.

That there is some abnormality in the sympathico-adrenal mechanism is suggested by the observation that patients displaying the syndrome of physical allergy do not seem to react to stress in an efficient manner.

In discussing the autonomic nervous system Cannon[25] stated that the ways have been revealed in which pain, cold, various emotional states and other conditions which excite autonomic discharges can induce or participate in pathologic processes. Thereby morbid phenomena which have long been regarded as mysterious have been explained in a reasonable way. Cannon[26] stated that he and his co-workers were able to show that muscular work, emotional excitement, asphyxia, low blood pressure, external cold, infection and hypoglycemia were accompanied by an extra secretion from the adrenal medulla and extra activity of the sympathico-adrenal system. Such services performed by the sympathico-adrenal system at times of unusual or critical need, Cannon has termed its "emergency" functions. Cannon showed that experimental animals deprived of the sympathico-adrenal system were normal so long as they were not subjected to stress.

White and Smithwick[27] stated that according to the evidence available the parasympathetic system never reacts with a diffuse discharge to "natural" stimuli. This suggests than an abnormality may be present in the vasomotor nervous system of persons who have physical allergy.

Selye[28] has stated that whenever the organism is placed in a critical situation it meets it with a typical tissue defense reaction. Selye felt that at least some of the symptoms of the alarm reaction are due to the liberation of histamine from various tissues of the body. He concluded from his study that one of the functions of the adrenal glands is to counteract or detoxify this metabolite. Exposure to variable surrounding temperatures and excessive fatigue are among the agents likely to act as alarming stimuli.

Kendall[29] stated that the changes in the distribution and excretion of inorganic ions and in carbohydrate metabolism which occur in adrenal deficiency are associated with the metabolic processes which involve fats and proteins. It is, therefore, not surprising to find a subnormal state in many physiologic processes in association with deficiency of the adrenal gland. The physiologic processes likely to show this subnormal state are the maintenance of blood pressure and body temperature and the resistance to stress, long continued muscular activity and toxic substances. Kendall stated that the known physiologic processes which are affected by the hormones of the adrenal cortex can be grouped in three divisions. These are: (1) the control of permeability and the transfer of inorganic ions and water between extracellular and intracellular phases and from blood to urine; (2) the activity of tissues with specialized functions, such as the liver, kidney, gastro-intestinal tract and muscles; after adrenalectomy these physiologic processes are disturbed in large part because of changes in permeability and in the distribution of water and inorganic ions, especially sodium and potassium; and (3) the activation of enzymes. As stated previously, increased permeability of the blood vessels is characteristic of all allergy whether of the physical or the antigen antibody type.

Sensitization. Duke[30] was of the opinion that specific hypersensitivity to physical agents could be the result of the development of a chemical substance in certain tissue. Following such sensitization, stimuli from the vasomotor system will release the chemical substance or substances to produce either general or local reactions.

The work of Lewis[12] on sensitization of skin to physical irritants, such as heat and cold, suggested the manner in which blood vessels might become sensitized. He also noted that such sensitization took place more readily in the presence of infection.

Danielopolu and Marcou[31] found that in an adrenalectomized animal after all traces of vasoconstrictor action of epinephrine had disappeared, if only a trace of epinephrine was injected into the animal, the blood vessels immediately regained their initial tonus.

Mechanism. The evidence in regard to intrinsic allergy which I have reviewed briefly is suggestive of a fundamental, inherited, localized, cellular abnormality resulting in some change in cellular permeability and concomitant release of histamine when the deficient cell is affected by stimuli which obtain little or no response from the

normal cell. The disorganization produced as a result of the stimulus is an injury response of the cell, causing release of histamine with subsequent vasodilatation and pain. There is evidence to suggest that the sympathetic and parasympathetic balance may not be normal, in addition to carrying stimuli to an abnormal cellular substratum.

GENERAL DESCRIPTION OF THE TYPE OF CASE AND THE PROBLEMS UNDER CONSIDERATION

I have considered intrinsic allergy at some length because, although well recognized by investigators, this concept of allergy has not yet received wide publicity and because in the practice of otology and rhinology the type of allergy we have to deal with is usually of the intrinsic type.

Allergies, affecting the nose, the paranasal sinuses and the ears, have plagued the otorhinologist since the inception of the specialty because of the associated infection and pain in the head.

Patients who have vertigo or pain in the head frequently consult the otorhinologist because the pain followed severe infection of the upper respiratory tract, frank suppurative sinusitis, or otitis media and mastoiditis although signs of such infection may have completely disappeared. More frequently the pain in the head is associated with nasal discharge which the patient may term "cold in the head" or "sinusitis," or with a chronically draining ear which the patient may believe indicates involvement of the mastoid process with impending intracranial complications.

The diagnosis of pain in the head associated with acute inflammation of the paranasal sinuses has never been the subject of controversy. The pain and tenderness are in the same place over one of the sinus walls; the pain is of the superficial type. The same may be said of acute mastoiditis. In chronic suppurative sinusitis and otitis pain in the head rarely occurs. The point most striking to me about uncomplicated suppurative otitis and sinusitis is that reference of the pain to a distance does not occur as far as I have observed.

In hyperplastic sinusitis, which is a condition in which symptoms and signs of vasomotor rhinitis and suppuration are intermingled, the picture is radically different. In such cases referred pains and bizarre headaches are frequent. It is in regard to this type of case that the controversy over the description and cause of the

headaches and other symptoms has arisen. It has arisen because the headache does not seem to have a close anatomic connection with the inflammatory condition in the nose or ear that is presumed to have caused it. The patients have difficulty in finding a simile to describe their subjective sensations. The pains tend to be referred. The distribution of pain seems impossible on the basis of the known distribution of the cutaneous sensory nerves.

Therapeutic relief by both surgical procedures and nerve blocking has been inconstant, usually futile. The patient and a series of physicians have become discouraged; the patient's condition has become worse. The majority of this group of patients make the self-diagnosis of "sinus headache." Since, as I have indicated previously, confusion does not arise in cases of frank suppuration, it seems apparent that the pain in the head is the result of allergy rather than of infection. In accord with this concept many persons can be found who do not have sinusitis or chronic aural suppuration but do have the bizarre distribution of pain in the head.

Until recently the idea of infection as the cause of headache has been so firmly implanted in rhinologists' minds, however, that whenever pain in the head was present and evidence of frank suppuration could not be found, the phenomenon was explained on the basis of nonsuppurative infection characterized by edema and polypoid formation. Vast numbers of patients who had hyperplastic sinusitis were subjected to operative procedures as a result of this hypothesis with such an obvious lack of success that the idea grew up among both physicians and the laity that sinusitis could not be cured by surgical procedures. The abolition of the false concept of nonsuppurative infection has permitted consideration of pain and vertigo just described for what they really are, manifestations of allergy, and in this way to clear up much of the confusion in regard to them.

Unfortunately, treatment by routine allergic methods has failed to be of help in many instances. It was not until these cases were recognized as primarily instances of physical or intrinsic allergy that progress was made in relieving the patient of the symptoms for which he needed relief.

SLUDER'S SYNDROME

Sluder[32, 33] (1908) was the first to recognize the essential unity of the group of symptoms under discussion, but unfortunately was one of the principal proponents of the nonsuppurative infection

theory. For the purpose of comparing the Sluder syndrome with the syndrome of "intrinsic allergy" which I will describe later, I have summarized the symptoms and signs of Sluder's syndrome from his monograph[34] as follows:

I. Background
   - A. Often more or less severe inflammatory disease before development of the syndrome
   - B. Vasomotor rhinitis, asthma and myalgia often associated with the headache (or head pain)

II. General symptoms
   - A. Pain—unilateral or bilateral: occurrence in acute exacerbations or "attacks" on a substratum of discomfort; "sense of muscular tension"; not otherwise described
     1. At root of nose
     2. In and about eye
     3. In upper jaw and teeth
     4. Extending backward to the temple and along the zygoma to the ear
     5. Extending down the posterior aspect of the mastoid to the neck
     6. Reaching backward from the mastoid to the occiput
     7. Extending to the shoulder, less often to the axilla and breast
     8. Occasionally extending to the shoulder and arm
     9. Referred to the vertex
   - B. Tenderness
     1. Severest at a point 5 cm. back of the tip of the mastoid
     2. Associated with stiffness in "the muscles of the neck" after an attack
   - C. Vasodilatation, flushing and sense of heat over the face

III. Symptoms referable to the ear
   - A. Tinnitus—character not described
   - B. Tubotympanitis (stuffy ears)
   - C. Vertigo—usually mild but at times comparable to true Ménière's syndrome

IV. Symptoms referable to the nose
   - A. Stuffiness (localization to one side not described)
   - B. Watery discharge
   - C. Itching

V. Symptoms referable to the mouth and throat

A. Glossodynia or burning of tongue (common)

B. Parageusia (rare)

C. Salivation (rare)

D. Pain on swallowing

E. Globus

F. Itching of hard palate and tonsillar pillars

VI. Symptoms referable to the eye

A. Conjunctival injection

B. Lacrimation

C. Either dilatation or narrowing of pupil

D. Widening or narrowing of palpebral fissure

E. Asthenopia

F. Photophobia—occasionally a sense of photophobia when no light is present

VII. Treatment

A. Cocainization of sphenopalatine region in nose for temporary relief of symptoms

B. Operation on "involved sinuses" as advocated by Sluder for more permanent relief

1. Frequent failure to give relief

While refinements and additions in observation and description have been made since Sluder's final monograph, the referred pains in the head and signs and symptoms associated with hyperplastic sinusitis and aural disease still appear to be those recognized by Sluder. One is amazed that in complete lack of knowledge of the physiologic and anatomic bases of these symptoms and signs he should have been able to perceive a fundamental unity.

SYNDROME OF INTRINSIC ALLERGY

For purposes of description I have termed an interrelated group of painful symptoms about the head "the syndrome of intrinsic allergy." To investigate the probable mechanism of the syndrome it can be broken down into four main divisions as Sluder's syndrome can be: namely into vasomotor rhinitis, Ménière's syndrome, myalgia and the vasodilating pain syndrome. These main divisions may occur singly or in combination. Even when the symptoms of one or

another of these divisions are so prominent as to seem to form a unique syndrome, careful investigation sometimes will show that many symptoms or signs of the other three divisions are also present. Table I prepared after clinical observation of more than 300 patients, I believe, will reveal the essential clinical unity of these four main divisions.

Vasomotor Rhinitis.—Lillie was one of the first rhinologists to insist that the treatment of rhinologic conditions should be based on physiology rather than anatomy. He recognized Sluder's syndrome as an entity but was dissatisfied with the results obtained by the surgical methods of treatment. He, therefore, asked Hansel, then a fellow in the Mayo Foundation to study this question. As a result Hansel wrote and published a thesis on vasomotor rhinitis.[35] Allergy later became Hansel's major preoccupation and in many papers summarized in his monograph,[36] he was able to demonstrate the allergic nature of vasomotor rhinitis. By reviewing 100 microscopic sections of material removed by Sluder in operations designed to relieve the Sluder syndrome he was able to show that[37] what Sluder thought a peculiar type of infection characterized by polypoid hyperplasia and sphenopalatine ganglion neuralgia was really allergy. In 66 of the 100 microscopic sections reviewed there was preponderant eosinophilic infiltration; the other slides revealed either normal mucosa or acute infection. Hansel's work has established at least the vasomotor rhinitis component of the Sluder syndrome on an allergic basis.

In our present work at the Mayo Clinic we make the diagnosis of perennial vasomotor rhinitis on the basis of a history of paroxysmal attacks of sneezing followed by nasal congestion and watery discharge from the nose, nearly always bilateral, together with the observation of a pale edematous mucosa, especially over the turbinates, covered with a watery or mucoid discharge. If, in addition to these findings, an abundant purulent secretion bathes polyps, the condition is diagnosed as hyperplastic sinusitis which is considered in this paper as being perennial vasomotor rhinitis with secondary sinusitis.

Ménière's Syndrome.—The diagnosis of Ménière's syndrome has been said to depend on sudden attacks of rotational dizziness or vertigo, tinnitus and deafness in the involved ear. Nystagmus is said to accompany the attack and to be rotatory or vertical with the quick component of the nystagmus either away from or toward the affected ear. There is no general agreement on this point, however. Nausea with cold sweat and vomiting is usually included. Most observers have thought that the condition is usually unilateral. Harris

and Moore,[38] however, stated that bilateral Ménière's syndrome is relatively common.

The cause of Ménière's syndrome is unknown. Crowe[39] gave the following reasons among others for suspecting that the peripheral system of semicircular canals and cochlea rather than the nerve or the vestibular nucleus was the seat of the disturbance. The vestibular symptoms of Ménière's syndrome differ only in intensity from those produced by the functional tests. The infrequency of vestibular symptoms from intracranial tumors argues against the disease being in the nerve or the central vestibular nucleus. The involvement of other cranial nerves in Ménière's syndrome is rare. Trauma of the vestibular nerve does not cause symptoms. Crowe was of the opinion that the strongest reasons for suspecting variable pressure and chemical changes in the membranous labyrinth as the cause of Ménière's syndrome were the fluctuating character of the deafness and the frequency and length of the remissions. Mygind and Dederding[40] pointed out that the symptoms could be controlled to some degree by dehydration. Furstenberg, Lashmet and Lathrop[41] stated that the balance between the sodium and potassium ions in the endolymph was concerned in the production of Ménière's syndrome and suggested a diet low in sodium ion supplemented with large doses of ammonium chloride. Talbott and Brown[42] felt that a high intake of potassium may be of more importance in controlling Ménière's syndrome than a low intake of sodium. In four cases studied during an attack of Ménière's syndrome an increased concentration of serum potassium and a decreased concentration of serum sodium were noted. These workers suggested the hypothesis that an increase in serum potassium indicates a loss of tissue potassium just as a high level of serum calcium accompanies a loss of calcium from the tissues. They suggested also that the depleted potassium in tissues can be restored and electrolyte equilibrium reestablished by the administration of additional potassium.[43]

Hallpike and Cairns[44] were the first to report the pathologic changes in two authenticated cases of Ménière's syndrome. They reported gross dilatation of the endolymph system affecting chiefly the scala media of the cochlea and the saccule. Degenerative changes were present in Corti's organ. They reported a conspicuous absence of inflammatory change. Hallpike and Wright[45] reported similar observations in another case. These pathologic findings have been confirmed by Lindsay[46] and others. These findings are in accord with the concept of an allergic etiology for Ménière's syndrome. Lindsay felt that the roles of allergy and vasomotor instability played a part

in the production of symptoms, although he stated that clinically there were strong contraindications to Ménière's syndrome being on an allergic basis, that allergic manifestations were rare in Ménière's syndrome and that attacks of vertigo were not common in allergy. Brown,[47] as evidence in opposition to Lindsay's opinion, found a history of "vasomotor imbalance" common in the family histories of patients suffering from Ménière's syndrome. Halle[48] pointed out the association of deafness, vertigo and nystagmus to myalgia as far back as 1911.

Shambaugh and Roberts[49] found that the detection of the allergic factor in Ménière's syndrome was most difficult owing to the unreliability of skin tests in this condition.

Atkinson[50] cited four cases of Ménière's syndrome reported by Dohlman[51] in which the etiologic factor was allergic and presented two cases of his own in which similar symptoms were relieved by elimination diets.

From my own observations I do not believe that Ménière's syndrome can be sharply divided from vertigos without deafness and tinnitus, as suggested by Atkinson. In my cases I see a gradual gradation of postural vertigo, that is, vertigo brought on by sudden change of position, up through tinnitus and vertigo without loss of hearing to the so-called true Ménière's syndrome. Many patients exhibiting the diagnostic triad of vertigo, tinnitus and deafness also will much more frequently have minor episodes on sudden change of position which are typical of so-called postural vertigo rather than of Ménière's syndrome. For the purpose of this paper, however, a diagnosis of Ménière's syndrome has rested on the presence of vertigo, tinnitus and loss of hearing in the affected ear or ears.

I believe sufficient evidence has been produced for the tentative classification of Ménière's syndrome as an allergy and as belonging to the larger syndrome of physical or intrinsic allergy as it involves the various structures of the head.

Myalgia.—Localized painful spots in muscles producing reference of pain in a distribution not that of the cutaneous nerves were first described by Froriep[52] in 1843. I have[53, 54] considered myalgia of the head and pharynx in other papers. One of the earliest and most enlightened discussions on myalgia[55] was held in the Senate Chamber of the State Capitol of Minnesota in St. Paul on June 19, 1877, by the Committee on the Practice of Medicine of the Minnesota State Medical Society. In the course of the discussion it was mentioned that the tissue changes produced by the "rheumatic

agency" were not observed in myalgia or "muscular rheumatism." The tissue changes in "rheumatism" had been pointed out by Virchow and Froriep[56] to be hyperemia, serous exudations, inflammatory proliferation of connective tissue and changes in the sarcolemma and neurilemma. These changes were found to be absent in myalgia. In the discussion by the Committee on the Practice of Medicine just mentioned myalgia was thought to be a metabolic disturbance in muscle, which possibly depended on the lack of elimination of lactic acid and produced an irritation of the nerve endings in the muscle. It was thought that this metabolic disturbance depended on the premature checking of blood flow to the muscle and as a consequence the requisite nutrient supplies were withheld, the interchanges that reduced irritant to inert products were impeded, and the irritant bodies were detained within the muscle. It also was stated that atmospheric pressure had a decided effect in precipitating myalgia and the effect of drafts and sudden cooling in bringing on attacks was pointed out. It was observed that the Chippewa Indians, especially the squaws, were very likely to have myalgia. For treatment, opium and its derivates and salts of potassium were recommended. The local measures advised were heat and massage. At this meeting too it was mentioned that salicylic acid and salicin, drugs that seem to be almost specific in the treatment of fibrositis, had no influence whatever on myalgia. Few of the later papers on myalgia have exhibited the knowledge of physiologic possibilities presented in this early discussion.

Sluder[57] stated that cases reported as myalgia in the literature were in fact instances of his syndrome.

Lange[58, 59] described myalgia under the title of "myogelosis." He found that macroscopic and microscopic examinations of tissue excised from the tender region did not reveal pathologic change. A number of observations demonstrated that myogelosis does not disappear during deep general anesthesia. The absence of pathologic change is consistent with an allergic etiology of myalgia. Lange concluded that myogelosis is not a circumscribed spasm of muscle fibers but is the result of an organic change probably colloid chemical in nature. Maliwa[60] presented evidence that myalgia occurred in combination with allergies. Sell[61] stated that muscles with a chiefly postural function contain more sarcoplasm than muscles for quick motion, and that those rich in sarcoplasm are the ones predisposed to myogelosis.

It remained for Lewis[12] and Kellgren[62-64] and associates, however, to establish myalgia on an experimental basis. They demon-

strated that the pain in myalgia is the same as that produced by exercising a muscle under ischemic conditions and that in myalgia the pain from a given muscle is felt over definite regions and seems to follow a segmental pattern. Lewis concluded that the pain from a given muscle is determined by a chemical or physiochemical stimulus developed in the muscle mass, which he called the factor "P" and that the process was rapidly reversible in the presence of oxygenated blood. Lewis found the pain from muscle to be "deep pain" which is not well localized, because impulses from visceral and parietal structures are recognized as pain and referred to the whole muscle segment rather than accurately localized. He stated that sharper localization than this had not been found useful in phylogenetic development. Lewis also has shown that skin may be sensitized to physical agents so that a further exposure to such agents will produce a reaction characterized by release of histamine. In this he anticipated the recent observations on immersion foot. Lewis was of the opinion that the process in muscle producing myalgia was similar to that which he had observed in the sensitization of skin.

In observing the mechanism of pain in the head produced by histamine, Pickering and Hess[65] suggested that the headache produced by the intravenous injection of histamine would be explained completely if the headache were due to stretching of a sensitive structure lying close to the meningeal arteries. Such stretching might result from swelling of the perivascular tissues or from widening of the meningeal arteries. They showed that while amyl nitrite will produce an expansion of the brain and a lowering of the blood pressure identical to that produced by histamine, it will not produce headache. It is known that histamine increases the permeability of the minute blood vessels which amyl nitrite has not been shown to do. An increase in vascular permeability might be a factor in producing headache if the pain were due to stretching of the perivascular structure from edema secondary to the increased permeability of the capillaries.

The studies of Lewis[12] and his co-workers lead to the hypothesis that in myalgia due to local sensitization of cells, local arteriolar vasoconstriction might lead to a local deficit in oxygen so that normal reversal of the catabolic process could not take place. Such failure would result in the piling up of enough of the P substance to produce pain. That certain vasodilating substances also are produced, possibly histamine, is suggested by the fact that in the exacerbation of severe pain called "attacks" the pain, at least at first, is pulsating. In the group of cases considered in this paper the diagnosis of myalgia

of the head or pharynx has been based on precipitation of a painful syndrome in the neck and head by sudden changes in temperature or barometric pressure. The pain was associated usually with unilateral tenderness and stiffness in certain groups of muscles, which had none of the characteristics of fibrositis (jelling, relief with mild exercise, relief from salicylates and tendency to recur in different locations) and with pain referred to the regions worked out by Cyriax.[66] The differential diagnosis from migraine and other conditions with which myalgia might be confused is covered in previous papers on myalgia of the head and myalgia of the pharynx.

Vasodilating Pain Syndrome.—Wolff[67] demonstrated that traction on, or dilatation of, the cranial arteries will produce pain but crushing or spasm will not.

Schumacher, Ray and Wolff[68] stated that the large arteries at the base of the brain, which include the internal carotid, the vertebral, the basilar artery and the proximal portions of their branches, are the primary sites of origin of pain in the head resulting from histamine, although the extracranial arteries are subsidiary sources of pain.

The work of Pool and Nason[69] suggests that the branches of the external carotid artery are adrenergic and the branches of the internal carotid artery are cholinergic. Thus epinephrine given intravenously, while relieving the pain of the vasodilating pain syndrome involving the extracranial vessels, would tend to accentuate the pain originating from intracranial vessels as in migraine.

Ray and Wolff[68, 70] described six basic mechanisms of headache, chief of which were dilatation and traction on the venous sinuses and intracranial and extracranial arteries. They stated that headache from intracranial disease is usually referred pain. They also found that the chief pathway for sensation of pain in the head was by way of the fifth cranial nerve but that the seventh and ninth cranial nerves and the first three cervical nerves also served as pathways.

Horton, MacLean and Craig[71] showed that a vasodilating headache may be produced by injections of histamine insufficient to provoke a histamine headache; the pain starts from thirty minutes to an hour after the injection of histamine. This delay suggests some intermediate mechanism by which the injection of histamine sets free, as Pickering and Hess suggested, after a latent period, the P substance of Lewis, possibly histamine, at the same time producing vasodilatation.

That parasympathetic overaction is the principal factor in both migraine and the vasodilating pain syndrome is indicated by the findings of Schumacher, Ray and Wolff[68] and of Horton, MacLean and Craig.[72] These studies also show that cervical sympathectomy has no effect in preventing the development of either migraine or vasodilating pain syndrome. Horton[73] also demonstrated that increased sweating, except for the circumoral sweat glands, a cholinergic phenomenon, accompanies the vasodilating pain syndrome. Schumacher and Wolff[74] showed that in migraine central vasoconstriction produces the prodromal scotomata. This might represent a preliminary action of the sympathetic nervous system followed by a powerful parasympathetic reaction. This suggests a mechanism, vasoconstriction followed by dilatation, by which not only the vasodilating pain syndrome but all the phenomena under consideration might be precipitated.

I have found that watery discharge from the nose in vasomotor rhinitis and the head pains of myalgia can be activated after a latent period, similar to that reported by Horton, MacLean and Craig in cases of histaminic cephalgia, by injections of histamine subcutaneously. That the effects of subcutaneous and intravenous injections of histamine are comparable is suggested by the studies of Kahn[1] on the rate of diffusion of histamine injected subcutaneously. I feel, therefore, that the difference between the histaminic cephalgia of Horton and the exacerbations of headache seen in myalgia, vasomotor rhinitis and hyperplastic sinusitis and Ménière's syndrome are differences in degree rather than in kind. This idea is further buttressed by the finding of Wolff, Hardy and Goodell[75] that if a painful stimulus produced by the concentrated thermal radiation from a 1,000 watt lamp was allowed to fall on the forehead during a histaminic headache, it was possible by increasing the intensity of the secondarily induced pain to reach a level that made the underlying headache imperceptible. These authors also found that pain sense had no spatial summation. Expressed more simply, this hypothesis is that the total effect of several intensities of pain acting simultaneously is equal not to their sum but merely to the intensity of the severest pain. It can be seen that an intense pain from one part of a stimulated vessel would overshadow the appreciation of pain stimuli from other parts. It can be observed in cases of conjoined myalgia and vasodilating head pains that the more severe the vasodilating headache becomes the less appreciation the patient has for subsidiary pains which are only noticeable when the pain is increasing or diminishing or if the vasodilating factor of the pain does not reach an intensity sufficient to abolish the appreciation of less severe pains.

Horton has demonstrated that histaminic cephalgia probably involves branches of the external carotid artery. It has been shown that ergotamine tartrate and epinephrine[69] contract the extracranial vessels and these drugs will relieve the pain of histaminic cephalgia. That the extracranial arteries are the chief source of pain in myalgia also is suggested by the fact that intravenous injection of epinephrine will relieve the pain. Relief of pain on injection of epinephrine also indicates a similarity between the vasodilating headache of the histaminic cephalgia type and the headache associated with the myalgia, and distinguishes both from migraine, because in the first two types of headaches the extracranial arteries are chiefly involved, while in the latter the intracranial vessels also may be a source of pain.[71]

In the cases considered as vasodilating pain in this paper the diagnosis rested on the presence of "visceral" pain in the head, sudden in onset, of a pulsating character unless extremely severe, usually unilateral but occasionally bilateral or midline. When the pain occurred in the anterior portion of the head, it usually was associated with lacrimation, conjunctival injection, swelling of the eyelids, congestion and discharge on the same side as the pain or on both sides if the pain was bilateral. The pain tended to appear frequently at night, would awaken the patient and was usually less severe if the patient stood upright than if he lay down. When the pain was present and often in intervals free of pain, the common carotid and its branches and the occipital artery were tender to pressure. Occlusion of the vessels tended to relieve the pain which flooded back when the pressure on the vessels was released. This type of pain in the head or headache has not been observed in any case in this series in which the patient was less than thirty years of age.

Diagnosis in 362 Cases Drawn from the Files.—Three hundred sixty-two case records drawn from the files were studied. In each case the painful symptoms about the head which I have now included in the syndrome of intrinsic allergy were present. The clinical diagnosis in this group of cases is given in Table 2.

In 173 cases a diagnosis of perennial vasomotor rhinitis only was made and in 77 vasomotor rhinitis was found to be associated with one or the other of the divisions of symptoms previously mentioned. I believe the grouping in Table 2 bears such a similarity to the syndrome proposed by Sluder that the syndrome of physical or intrinsic allergy and Sluder's syndrome may be considered identical.

On reviewing the records of the patients studied in preparing this report, I noted that patients who had perennial vasomotor rhin-

itis almost invariably stated that sudden change of temperature affecting the surface of the skin was a precipitating factor in their complaint. In myalgia of the head and pharynx a history of the appearance of the complaint following fever or exposure to extreme cold was often obtained. In myalgia a history of exacerbation of symptoms following sudden changes of barometric pressure, such as in approaching storms, as well as the precipitating effect of sudden chilling of the skin was obtained. In the vasodilating pain syndrome and Ménière's syndrome, I have rarely been able to obtain a history of precipitation by external physical agents except when vasomotor rhinitis or myalgia was associated.

It is this definite, fairly frequent association, however, which has led me to include the vasodilating pain syndrome and Ménière's syndrome in the syndrome of physical allergy.

REVIEW OF TYPES OF TREATMENT EMPLOYED FOR MEMBERS OF THE SYNDROME OF INTRINSIC ALLERGY IN GENERAL

The first treatment suggested for a member of this symptom complex was that of Sluder in 1908 who anesthetized the sphenopalatine ganglion to relieve neuralgia of the sphenopalatine ganglion. He later suggested injection of the sphenopalatine ganglion with phenol. Lillie and Childrey[76] have shown that cauterization of the filaments emerging from the sphenopalatine ganglion will destroy them and, if the ganglion is superficially placed, it actually will destroy the sphenopalatine ganglion itself. They reported relief of symptoms after application of silver nitrate. Sewall[77] suggested actual removal of the sphenopalatine ganglion.

In a recent review of neuralgia of the sphenopalatine ganglion Eagle[78] stated that the condition is characterized by unilateral pain in the face which never extends above the level of the ear. The pain is lancinating and continuous. Usually there are two main points of maximal pain, one in the region of the orbit and the other just posterior to the mastoid process of the temporal bone. Eagle mentioned reference of pain, the occurrence of vasomotor changes on the homolateral side of the nose and tinnitus on the homolateral side with vertigo. Eagle expressed the belief that nasal contact was the usual cause of sphenopalatine neuralgia and found that 80 per cent of 159 patients required some type of intranasal correction. He stated that all except one of the 53 patients operated on were relieved of their symptoms. Martin,[79] discussing the same syndrome, stated that the pain was transmitted along the arterial tree, through

the carotid sheath to the cervical and thoracic portions of the spinal cord. He reported a case in which temporary relief was obtained by resection of the sphenopalatine ganglion and concluded that resection of the sphenopalatine ganglion did not give permanent relief to neuralgia of the sphenopalatine ganglion because the efferent and afferent nerves of the periarterial plexus took over the function of the ablated nerves. This was indicated by the return of the vasomotor response.

In the cases I have studied I have found that cocainization of the sphenopalatine ganglion will relieve temporarily many of the pains that occur about the face and forehead, but that it usually does not relieve pain in the temples and cranial regions.

A few cases have been found in which submucous resection was necessary to get rid of a "trigger" area in the nose, but in these subsequent treatment with nicotinic acid or histamine was required to relieve the pain. At the Mayo Clinic we have not been able to equal Eagle's record in relieving sphenopalatine neuralgia by submucous resection alone, but we can confirm his statement that in certain instances such a result has been secured.

Various types of treatment have been suggested for Ménière's syndrome. Although most of these were mentioned in connection with the description of Ménière's syndrome, I shall list them briefly here: administration of potassium to control electrolyte equilibrium; the Furstenberg regimen and general measures including elimination of foci of infection.

Simonton[80] noted relief from vertigo by the Furstenberg regimen in 34 per cent of cases, partial relief in 28 per cent and no relief in 38 per cent of the cases studied. Walsh and Adson[81] found that 36 per cent of the patients in their series were relieved of vertigo on the Furstenberg regimen, 26 per cent were improved and about 39 per cent showed no improvement. They found that on administration of potassium nitrate vertigo disappeared in 31 per cent of their cases, improvement occurred in 46 per cent and in 23 per cent no improvement occurred. Of 22 patients treated by other general measures, including elimination of foci of infection, relief or improvement was obtained in 78 per cent, but in this group the improvement did not immediately follow treatment. The statistics of Simonton and of Walsh and Adson in regard to medical treatment are based on a period of observation of from six to eight months. The results of Walsh and Adson indicate that "general measures with removal of foci" gave better eventual results than medical treatment.

This unusual result probably depends on the tendency toward spontaneous remission in Ménière's syndrome.

House Dust.—Hansel[82] found that in treating allergies of the nose with house dust that satisfactory results were dependent on optimal dosage. He observed that poor results usually were due to starting with too strong a dose or overstepping tolerance with a dosage which was too high. He was of the opinion that dust extracts contain some specific unknown antigen. He, like Shambaugh,[49] was dissatisfied with the usual method of treating the allergy in many of these cases except when house dust was used. In the light of the work of Selye in regard to the alarm reaction it would seem possible that results of treatment with house dust depend on the irritant action of the dust rather than on the action of an unknown antigen.

Since therapeutic dosages of dust were of the same order as those found effective in histamine therapy, it was thought the "unknown antigen" might be histamine. Code demonstrated that Hansel's dust extracts do not contain histamine but I have found that the general principles outlined by Hansel as applying to dust therapy also apply to histamine and nicotinic acid therapy.

Histamine.—In 1929, Horton and Brown[83] suggested that since the patient who is sensitive to cold is also sensitive to a chemical substance formed in the tissues of the body, autodesensitization would be possible. Two procedures were considered: (1) desensitization by repeated injections of histamine over long periods in doses so minute that they would be insufficient to produce a systemic effect and gradual increase in the doses, and (2) autodesensitization by daily immersion of the hands in cold water of decreasing temperature for increasing periods. At that time Horton and Brown chose the second procedure.

The first use of histamine in the group of cases of intrinsic allergy of the ear, nose and throat was apparently made by Deutsch, who treated myalgia with histamine iontophoresis; he reported his results in 1931.[84] He based this treatment on the work of Lewis, von Trendelenberg, Ebblicke and Török who suggested that anoxemia secondary to vascular spasm was responsible for the accumulation of a product of metabolism which produced the local symptoms. Deutsch, therefore, introduced histamine into the involved muscles with favorable clinical results.

In 1939, Horton, MacLean and Craig[71] reported the use of histamine injection for patients with vasodilating pain syndromes in the

head. This suggestion of Horton and Brown[83] which has been carried out in many conditions since 1929 opened up the first hopeful new line of treatment of the conditions under consideration that had been discovered for many years.

In Alexander's[85] experience neither histamine nor histaminase had any value in the treatment of extrinsic allergy in which the cause was a specific hypersensitiveness, but in other conditions, such as urticaria, or especially, intrinsic allergy, histaminase may be helpful.

Nicotinic Acid.—The use of nicotinic acid in the syndrome under consideration was first suggested by Harris and Moore[38] for Ménière's syndrome in 1940. They adopted its use because a large percentage of pellagrins have rotational vertigo. They had tried thiamine chloride first without benefit. They then added nicotinic acid, 250 mg. daily, with entire relief of the symptoms of 17 out of 20 patients treated. This observation awakened considerable interest in the use of this drug in Ménière's syndrome and was followed by several papers by Atkinson[86-88] who used essentially the same treatment.

Aring and his co-workers[89] showed that nicotinic acid and quinine nicotinate administered intravenously increased the intracranial blood flow for periods lasting from 20 to 60 minutes. This effect paralleled the flushing of the skin. Nicotinic acid amide was found to have no significant effect on the total intracranial blood flow.

Unna[90] found the toxic dose of nicotinic acid in mice and rats to be 4 to 5 gm. per kilogram of body weight by subcutaneous administration and 5 to 7 gm. per kilogram by oral administration. The prolonged oral administration of 2 gm. a day of nicotinate of sodium per kilogram of body weight to rats, chickens and dogs during a two months' period did not produce any symptoms of toxemia nor pathologic alterations in any organ. Moore[91] gave nicotinic acid in doses of 250 grains (16 gm.) a day to patients suffering from multiple sclerosis for periods up to a year without ill effect.

TRIAL OF THE SKIN TEST WITH HISTAMINE AS AN AID IN SELECTING TREATMENT IN CASES OF THE PHYSICAL ALLERGY SYNDROME

Atkinson felt that since there was evidence to show that the manifestations of allergy were due to the release of histamine, or a histamine-like substance, that intradermal testing with histamine

could divide patients who had Ménière's syndrome into a group that was sensitive to histamine and another larger group that was insensitive to histamine. He cited the previous report of Dzsynich and Gallé as to the use of histamine as a general indicator of allergy. Dzsynich and Gallé injected 0.001 and 0.00001 mg. of histamine in 0.1 cc. of saline solution beneath the skin of the forearm and reported that the test was positive for allergy when a wheal of 1 cm. and a surrounding flare of 2 cm. appeared. In his original paper Atkinson stated that he injected 0.01 mg. of histamine intradermally which I assumed to be histamine base. In a second paper published in 1942 Atkinson[92] apparently continued to follow this same technic as his sole statement as to method was that the technic previously described should be followed closely to secure good results. In an article published in 1943[88] he stated that an intradermal test is performed by injection of 0.0057 mg. of histamine base (0.01 mg. of histamine dihydrochloride). If this latter dose is taken as the one used by Atkinson, it can be seen that the dosage used in the cases on which this paper is based contained 0.0043 mg. more histamine base than that proposed by Atkinson in his third paper but it is assumed that this variation could have little effect on the result. The work reported in this paper was done before the correction in Atkinson's third paper was obtained.

Atkinson apparently disregarded the area of erythema surrounding the wheal and based his conclusions as to sensitivity to histamine on the presence or absence of "pseudopods" from the wheal. Lewis and his colleagues[93] described the skin reaction to histamine as a "triple response" consisting of vasodilatation of the small arteries at the site of the injection, a diffuse flare and an area of local edema or wheal which appeared over the small area exhibiting vasodilatation. They showed that the vasodilatation and wheal are due to direct action on the capillary wall. The flare, however, is caused by a local axone nerve reflex. Local vasodilatation and wheal formation depend on the direct stimulation of the cells of the vessel walls by the irritating agent. The flare occurs only in sensitized skin and is thought to be a specific response to histamine. Atkinson paid but little attention to this in his test, however, and depended on the production of pseudopods which Lewis has shown to be due to the distribution of the local lymphatics.

Before proceeding with the treatment of patients with either histamine or nicotinic acid it was thought suitable to test the validity of Atkinson's hypothesis in cases without physical allergy and the whole group of cases of the physical allergy syndrome.

Cases without Physical Allergy.—Fifteen patients who gave no history of physical allergy were subjected to the intracutaneous injection of histamine diphosphate. The dose was 0.01 cc. of a 1:1,000 dilution of histamine base. Negative results (no pseudopod formation) were obtained in eight cases; positive results (one or more pseudopods extending at least 1 cm. from the edge of the wheal) were obtained in seven cases. One patient who had a strongly positive response to Atkinson's test with two large pseudopods, extending 4 cm. from the edge of the wheal, and three medium sized pseudopods, extending 2 cm. from the edge of the wheal, was found to have a brain tumor. Another was found to have a duodenal ulcer on roentgenologic examination. The general physical examinations of the other five patients gave essentially negative results.

Cases of Physical Allergy.—Fifty-five patients whose conditions had been diagnosed as the physical allergy syndrome were then subjected to the Atkinson test and subsequently treated with either histamine or nicotinic acid without regard to the results shown on test. The results of the test and of treatment are given in Table 3. The result of treatment was considered good when complete or nearly complete relief of symptoms was maintained for three months or more and fair when only 50 to 90 per cent relief was maintained for three months or more.

In a further trial of whether skin tests with histamine were an aid in selecting treatment, 55 patients whose condition had been diagnosed as physical allergy were subjected to intracutaneous tests with histamine diphosphate; for these 0.02 cc. of a 1:50,000 dilution of histamine base was employed (Table 4). The criterion for a positive test was a flare extending more than 1 cm. beyond the edge of the wheal in all directions. The tests were all made in a closed room at a temperature between 75° and 80° F. The criteria as to results of treatment were the same as in the previous tests (Table 4).

Comment.—The following conclusions were drawn from these tests: 1. There does not seem to be any agreement between the results of skin testing with histamine and the results of treatment. 2. Skin tests with histamine seem valueless both for diagnosis and as a guide to therapy. Therefore, no further skin testing with histamine was done. 3. These findings do not bear out Atkinson's contention that vasospastic and vasodilating types of allergy exist, since the response in cases of all types in the physical allergy syndrome was equally good to treatment with either histamine or nicotinic acid.

TREATMENT OF MY SERIES OF CASES

The cases of the syndrome of intrinsic allergy about which I am reporting the results of treatment do not correspond to the 362 cases which were drawn from the files for a study of diagnosis and symptoms when I was formulating my impressions about the interrelated group of symptoms which I have now termed "the syndrome of intrinsic allergy." The treatment series is in general a later group of cases although there may be some overlapping. The treatment series consists of 327 cases in which nicotinic acid or histamine or both were employed. Thirty-two cases in which nicotinic acid amide, the extract of whole adrenal cortex or epinephrine was given and 15 cases in which Kendall's extract of whole adrenal cortex and epinephrine were used are considered in addition.

Method of Treatment with Histamine.—The solution of histamine diphosphate used was equivalent to a 1:50,000 dilution of histamine base. After trial with various dosages, it was found that 0.1 cc. of this solution, if given subcutaneously, would not precipitate symptoms in the majority of cases of the physical or intrinsic allergy syndrome. In the few cases in which exacerbation of symptoms was likely to be produced by this dosage, injections were started with 0.1 cc. of 1:100,000 dilution of histamine base or if necessary 0.1 cc. of 1:200,000 dilution. No patients were found who had an untoward reaction to the dilution last mentioned.

Following the initial dose patients received subcutaneous injections twice daily; the quantity of fluid injected whatever the dilution selected, was increased each time by 0.1 cc. until symptoms disappeared or an objective return toward normal in the appearance of the nasal mucosa in cases of perennial vasomotor rhinitis was observed. The dosage which would maintain the patient symptom free was termed the "optimal dosage" following the nomenclature established by Hansel in the treatment of vasomotor rhinitis with dust extracts. It was found that a small increase of the dose beyond that producing the initial symptomatic relief would result in a reappearance of symptoms and each further increase would produce marked increase in the severity of the symptoms.

When the optimal dosage of the twice daily injections was established, half the total daily dose was given once daily to see if improvement was maintained. It often was found necessary to increase the dose slightly to continue the patient symptom free. This daily dose was termed "the maintenance dose" and was found to vary between 0.1 cc. of a 1:50,000 dilution of histamine base and 0.1 cc.

of a 1:1,000 dilution of histamine base. The majority of patients, however, were maintained on 0.5 cc. of a 1:10,000 dilution of histamine base. An attempt was made in many cases to reduce the number of injections to either three or four weekly but such reduction was usually followed by recurrence of symptoms in a short time. The patients were advised, therefore, to continue the maintenance dose for six months and then discontinue treatment. In most instances in which this schedule was carried out, the original symptoms reappeared after a short time and either resumption of the daily injection of histamine or change to nicotinic acid by mouth was found necessary.

Method of Treatment with Nicotinic Acid.—It was found that most patients could tolerate an initial dose of 25 mg. of nicotinic acid (niacin) subcutaneously. In a few instances this dosage produced a marked increase in symptoms after delay of from fifteen minutes to half an hour. In several cases in addition to generalized flushing of the skin, abdominal cramps and involuntary movement of the bowels occurred. In a few cases treatment had to be started with doses as low as 1 mg. of nicotinic acid given subcutaneously. In the average case, however, twice daily injections could be given, with an increase of 25 mg. on each injection, until a dose of 100 mg. was reached. In the average case 100 mg. of nicotinic acid injected once daily was found to be an adequate maintenance dose. In a few instances the optimal dose was found to be as low as 12.5 mg. daily. An increase above this level resulted in reappearance of symptoms. In several other instances, notably when vasodilating pain of the head was the chief presenting symptom, dosages of as much as 200 mg. daily had to be maintained. The cases in which deviation from a daily dose of 100 mg. was necessary were relatively few. For this reason, therefore, nicotinic acid is a more useful therapeutic agent than histamine diphosphate.

Patients were advised to maintain on a daily dose of 100 mg. of nicotinic acid subcutaneously (when this was the optimal and maintenance dose) for three months. At the termination of this period they were advised to take by mouth 100 mg. of nicotinic acid night and morning as it was found that nicotinic acid by mouth was less effective in maintaining symptomatic relief than when it was administered subcutaneously. Patients were instructed to continue oral administration for three months and then attempt to discontinue treatment. In a few instances symptoms have not reappeared after discontinuing treatment for periods of three months but in most cases oral treatment has had to be continued.

Results of Treatment with Histamine or Nicotinic Acid or Both.—Results of treatment in 327 cases are summarized in Table 5. Criteria for good and fair results and failure of treatment were the same as those employed in the trial of treatment in association with the cutaneous test with histamine. Various comments will be made also on details of the findings.

Perennial Vasomotor Rhinitis (Table 5).—In one case in which a good result was obtained in the treatment of vasomotor rhinitis with histamine and in another with nicotinic acid the associated asthma was not affected. In three of the 18 cases in which treatment with histamine was considered a failure, relief was obtained for two weeks, in one case for one week and in one for six weeks; in one case exacerbation of asthma was responsible for failure of treatment. In one case in which fair results were obtained from treatment with nicotinic acid septal deflection probably accounted for lack of relief.

Various cases in which treatment with nicotinic acid was classed as a failure should be mentioned briefly. In two of these cases in which immunologic allergy was associated, symptoms became severe when preseasonal "desensitization" was attempted after two months' relief. In one case of chronic ulcerative colitis, nicotinic acid seemed to increase gastro-intestinal symptoms. In two cases asthma was precipitated by injection of as little as 1 mg. of niacin. In one case after two weeks' relief, a violent attack of vasomotor rhinitis was precipitated half an hour after injection (after initial flush of skin had disappeared). In two cases of immunologic allergy and positive skin tests relief was complete for one month. In three cases asthma developed after one month. The asthma, however, disappeared on cessation of treatment.

When both nicotinic acid and histamine were employed, success was obtained with nicotinic acid after failure with histamine in three cases and with histamine after failure with nicotinic acid in one case; 50 per cent relief was obtained with histamine after failure with nicotinic acid and histamine separately but complete relief on use of both together in two cases. Complete failure resulted from treatment with nicotinic acid and then with histamine in six cases. Simultaneous treatment with both was not tried in these cases. In one case failure resulted from histamine and an apparently good result was obtained for one month from niacin. Treatment in this case was terminated because of development of dermatitis of the hands. The dermatitis disappeared after treatment was stopped.

In another case an apparently good result with histamine disappeared after two weeks. With niacin the patient was relieved for two months before treatment had to be stopped because of the development of asthma, which disappeared on termination of treatment.

Perennial Vasomotor Rhinitis and Ménière's Syndrome With or Without Myalgia of the Head (Table 5).—One of the patients who had symptoms of both perennial vasomotor rhinitis and Ménière's syndrome was given histamine both intravenously and subcutaneously without relief. Relief was obtained subsequently with niacin.

One of the patients who had vasomotor rhinitis, Ménière's syndrome and myalgia was completely relieved of all symptoms on subcutaneous treatment with histamine except at menses when a major attack lasting several hours would occur. Treatment with histamine, therefore, was classed as a failure. The patient subsequently was relieved completely by niacin.

Ménière's Syndrome Only or Associated with Exacerbation of Myalgia (Table 5).—One of the patients who had Ménière's syndrome associated with exacerbation of myalgia obtained relief of symptoms for two months on treatment with histamine, then had a recurrence. This patient was subsequently relieved by nicotinic acid. Another patient who was having daily symptoms obtained complete relief on treatment with nicotinic acid for one month before recurrence.

Fair results (50 per cent relief of symptoms for three months or more) were obtained in three cases of Ménière's syndrome only on treatment with nicotinic acid. After two months relief from 100 mg. of nicotinic acid daily, vertigo with abdominal cramps recurred in one case. This patient was completely relieved by reducing the dose of nicotinic acid to 50 mg. daily and supplementing it with 10 grains (0.65 gm.) of potassium iodide with meals. In the one case in which failure from treatment with nicotinic acid occurred the patient first improved definitely for one month.

Vasodilating Pain Syndrome (Table 5).—Subcutaneous administration of histamine was considered a failure in three cases. One of these patients obtained relief for one month. One patient was subsequently relieved by nicotinic acid, and one was not.

Nicotinic acid given subcutaneously produced good results in three cases. Doses of 200 mg. of nicotinic acid daily were required in one case before relief was secured. In one of the three cases in which subcutaneous administration of nicotinic acid was considered

a failure, histamine given subcutaneously also failed to give relief. The patient was completely symptom free while receiving 5 cc. of the extract of the whole adrenal cortex as prepared by E. C. Kendall plus 1 cc. of 1:50,000 solution of epinephrine.

Vasodilating Pain Syndrome Associated with Perennial Vasomotor Rhinitis (Table 5).—Histamine given subcutaneously failed to give relief in three cases. In one of these cases intravenous treatment with histamine also failed. In two of the cases in which treatment with histamine was classed as a failure, relief for two weeks was followed by recurrence. Nicotinic acid gave complete relief for one month in the one in which this type of treatment was considered a failure.

Vasodilating Pain Syndrome Associated with Exacerbations of Myalgia of the Head (Table 5).—Of the two patients for whom subcutaneous administration of histamine was considered a failure, one was relieved of symptoms for six weeks by this means and the other obtained no relief and also failed to obtain relief from niacin. In the one case in which treatment with niacin was considered a failure treatment had to be stopped because of nausea and vomiting following injection.

Vasodilating Pain Associated with Exacerbation of Myalgia of the Head and Perennial Vasomotor Rhinitis (Table 5).—In two of the three cases in which treatment with histamine was considered a failure symptomatic relief for two weeks and in one case for one week was obtained.

Myalgia of the Head Associated with Perennial Vasomotor Rhinitis (Table 5).—In one of the three cases in which histamine administered intravenously failed to give relief, 0.1 cc. of 1:100,000 solution of histamine base produced a marked increase in symptoms ten minutes after injection on three occasions. No flushing of the skin could be observed.

In one of the 16 cases in which nicotinic acid given subcutaneously produced good results histamine given intravenously had produced marked increase in headache one hour after injection. In both cases in which treatment with nicotinic acid was a failure subcutaneous injection of histamine also failed to give relief.

Myalgia of the Head (Table 5).—Histamine given subcutaneously failed to give lasting relief in four cases, in one of which, however, symptoms were relieved for one month. In one of the 13 cases in which subcutaneous injection of nicotinic acid gave good results

abdominal cramps were produced by 100 mg. doses but relief was maintained by 50 mg. daily; in two of the six cases in which this type of treatment was considered a failure relief of symptoms was obtained for two weeks.

Comment.—It was concluded from the results obtained that histamine and nicotinic acid were equally effective in the treatment of vasomotor rhinitis. The good results reported by Harris and Moore in the treatment of Ménière's syndrome with nicotinic acid were confirmed. From the small number of cases in the series in which Ménière's syndrome was the only complaint no comparison could be made between the effectiveness of histamine and nicotinic acid in relieving symptoms.

The only conclusion that may be drawn is that both histamine and nicotinic acid administered as described herein are relatively useful drugs in treating the physical allergy syndrome. From the results obtained one may conclude that the effect is obtained by fundamentally the same biologic mechanism. The only contrary evidence obtained was in the two cases in which good results were obtained by the use of nicotinic acid and histamine in conjunction after each had failed to produce a good result when used separately. I am unable to explain this observation.

Treatment with Nicotinic Acid Amide.—An attempt was made to see if treatment with nicotinic acid amide would give results similar to those obtained with niacin (nicotinic acid). Sixteen patients were treated.

To this group 100 mg. of nicotinic acid amide were given subcutaneously daily for five days. The group consisted of five cases of vasomotor rhinitis, ten of myalgia, and one of vasodilating pain. No improvement in any of these cases was obtained with this treatment.

In a successful result of the treatment of vasomotor rhinitis with nicotinic acid, objective changes in the nasal mucosa are often observable within 24 hours after the optimal dose is reached. The results are somewhat slower in cases of myalgia and vasodilating pain of the head but in a successful case improvement in symptoms always occurs within 72 hours of reaching the optimal dose. The lack of response in the 11 cases of vasomotor rhinitis to nicotinic acid amide (niacin amide) suggests that the improvement is associated with the vasodilating effect of nicotinic acid which is absent in the nicotinic acid amide.

The number of patients treated is too small for the results to be conclusive but it seems improbable that nicotinic acid amide, given in doses comparable to those which had proved effective for nicotinic acid, could have failed to produce a single favorable response if nicotinic acid amide were of benefit in this type of case or if the benefit obtained by administration of nicotinic acid were purely psychogenic.

Treatment with the Extract of the Adrenal Cortex.—Because a review of the literature suggested that the abnormal cells involved in the physical allergy syndrome behaved as though they were deprived of cortical hormone Dr. E. C. Kendall kindly supplied me with his extract of the whole cortical substance for clinical trial. A preliminary report of the results obtained is appended.

Seven patients who had vasomotor rhinitis were given 5 cc. of the extract of whole adrenal cortex twice daily for three days without provoking any observable response. This is in accord with the observations of Prickman and Koelsche[94] in the treatment of asthma. Nine patients who had vasomotor rhinitis were given twice daily injections of 0.1 cc. of a 1:10,000 dilution of epinephrine for three days. Eight presented no objective or subjective change in the nasal signs or symptoms. One patient who had a peculiar combination of vasomotor rhinitis and migraine secured both objective and subjective relief from epinephrine and was subsequently maintained symptom free with nicotinic acid.

Fifteen patients with vasomotor rhinitis were given 5 cc. of Kendall's extract of whole adrenal cortex plus 0.1 cc. of a 1:10,000 dilution of epinephrine twice daily. Marked diminution in the swelling of the nasal mucosa together with definite return toward the normal pink color of the membranes was observed in 14 of these patients. There was also a marked reduction in the symptoms of nasal congestion, sneezing and watery discharge.

These results suggest both that there is a synergistic action between the hormones of the adrenal cortex and medulla and that an insufficient supply of these hormones may be present in the cells reacting in the physical allergy syndrome.

Control Series.—The 16 cases in which nicotinic acid amide was used and the 15 cases in which injections of adrenal cortical extract or epinephrine were used without effect give a control series of 31 cases which may be compared to the group receiving either histamine or nicotinic acid.

COMMENT

If the four symptom complexes presented in this paper are assumed to be representatives of an integrated group of symptoms, having as a basis physical allergy of the head, the facts gleaned from the study of one of the members of the larger symptom complex then may be correlated with the other members. If this assumption is correct, then the control of water and electrolyte balance should be of some value in the treatment of vasomotor rhinitis, myalgia and the vasodilating pain syndrome as it is in Ménière's syndrome. The importance of the control of permeability and the transfer of inorganic ions and water has been demonstrated in the case of Ménière's syndrome, by Mygind and Dederding,[40] Furstenberg, Lashmet and Lathrop[41] and Talbott and Brown[42, 43] and others. It does not seem unreasonable to assume that the disturbances of ionic balance and permeability which have been demonstrated for one member of the physical or intrinsic allergy complex are present in other members of this group of conditions since symptoms and signs which seem to depend on disturbances of metabolism and permeability of cell membranes are found in the entire group. That this is true is suggested by the empiric use of potassium salts in these conditions for years.

It can be assumed that sensitization to physical agents may take place and may occur more readily in the presence of infection. The clinical history, especially in cases in which myalgia of the head and vasodilating pain syndrome have followed extreme exposure to cold or have developed during an acute or chronic infection, reinforces the evidence for association of infection with sensitization. The good results obtained occasionally in these disorders by elimination of foci of infection may be on the basis of the elimination of a sensitizing factor.

These factors suggest that better clinical results might be obtained by combining several methods of therapy, such as the giving of potassium salts and dehydrating agents in combination with histamine or nicotinic acid. This should be the subject of further clinical investigation.

SUMMARY AND CONCLUSIONS

In this paper I have shown that many of the head pains associated with nasal and aural diseases are on the basis of a physical allergy rather than being due to the infection syndrome.

In considering vasomotor rhinitis, Ménière's syndrome, myalgia of the head and the vasodilating pain syndrome as interrelated com-

ponents of a syndrome of physical allergy it is necessary to show that they are interrelated, that they are allergic, and that the type of allergy involved is the intrinsic or physical type. This I feel has been done.

The allergic nature of perennial vasomotor rhinitis has been demonstrated so often that it now is generally accepted. The lack of relief secured by the usual allergic treatment seems to lie in the failure to distinguish between immunologic and intrinsic allergy. Most recent students of Ménière's syndrome feel that in all probability the disease has an allergic background. They may have been confused by the fact that clinically significant skin tests have not been obtained in cases of Ménière's syndrome and because other allergic manifestations were not found. This confusion seems to have arisen because of the existence of two symptom complexes with many similar symptoms, but with differing biologic mechanisms; ordinary allergy of the immunologic (antigen antibody) type and intrinsic or physical allergy in which an antigen antibody type of reaction does not seem to be present. Many of the difficulties in considering Ménière's syndrome as an allergy are cleared up by considering it as frequently an example of "intrinsic" allergy. The statement that Ménière's syndrome is not associated with other manifestations of physical allergy I feel to be incorrect, and I believe that further careful observation will substantiate this.

Myalgia and the vasodilating pain syndrome have not won general acceptance as "manifestations" of allergy and therefore I have presented at some length the evidence from the literature tending to substantiate this viewpoint as well as that in regard to the confusing pain reference in these conditions. I have also reviewed the work of Lewis on the sensitization of tissues to physical agents, a possible mechanism through which sympathetic stimuli, both objective and subjective, can set off a train of allergic symptoms. The response of these conditions to treatment both with histamine and nicotinic acid as well as their definite association with perennial vasomotor rhinitis are additional reasons for considering myalgia and vasodilating pain syndrome as allergic phenomena.

The interrelation of the symptoms of the intrinsic allergy complex is well brought out by the occurrence of vasomotor rhinitis in the vasodilating pain syndrome. Its presence is considered one of the diagnostic features of the condition. The simultaneous occurrence of vasomotor rhinitis and myalgia is also common. Halle and his associates have emphasized the association of Ménière's syndrome

and myalgia of the head. I have observed four patients who have had all four components of the "intrinsic allergy" syndrome; these were precipitated synchronously by the same physical stimulus, relieved by the same treatment and precipitable by appropriate doses of histamine and nicotinic acid. I believe that further clinical observation will confirm the interrelation of the four components of the "intrinsic allergy" syndrome, namely, vasomotor rhinitis, Ménière's syndrome, myalgia of the head and the vasodilating pain syndrome. The evidence has also been given which supports the idea that so-called histaminic cephalgia is not unique but is merely an outstanding example of the vasodilating pain syndrome.

I cannot refrain from quoting as a conclusion to this paper the remarks of that pioneer Minnesota physician, J. E. Finch, made at the "Symposium on Myalgia" before the Minnesota State Medical Association in 1877:

> To collect facts and endeavor to discover the principles which underlie and determine them is the noblest of mental effort. This is called theorizing and is particularly distasteful to some persons who seem to forget that without theory there is no science. In the theory here submitted, which is quite different from anything we have seen on the subject, we have endeavored to make use of facts or well recognized principles with the introduction of as little pure hypothesis as possible. We are not so vain as to pretend that it is complete in every aspect, but even if our reasoning be unsound and our conclusions fallacious, some of our brethren, while pointing out our fallacy, may alight upon the truth and thus our effort will not have been in vain.

I wish to acknowledge the advice and help which Dr. C. F. Code gave me in this study.

MAYO CLINIC.

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TABLE 1

COMPARISON OF SYMPTOMS OF VARIOUS COMPONENTS OF SLUDER'S SYNDROME WITH THE SYMPTOMS OF THE VARIOUS COMPONENTS OF THE SYNDROME OF INTRINSIC ALLERGY

CONDITIONS	SLUDER'S SYNDROME	VASCULAR HEADACHE, HISTAMINIC CEPHALGIA	MYALGIA OF HEAD	VASOMOTOR RHINITIS	HYPERPLASTIC SINUSITIS*	MENIERE'S SYNDROME
Background of sensitivity to cold	Present	Occasional	Present	Present	Present	Present
			PAIN			
Underlying muscular tenderness		Occasional	Characteristic	Occasional	Frequent	Often present
Unilateral or bilateral	Unilateral but may shift sides	Usually unilateral	Unilateral usually; may be bilateral	Occasional; unilateral or bilateral	Unilateral but may shift sides	Usually unilateral
Onset and termination	Sudden or slow	Sudden	Both slow	Slow	Usually slow, may be sudden	Same as myalgia
Character	Somatico-visceral	Somatico-visceral	Somatico-visceral	Somatico-visceral	Somatico-visceral	Same as myalgia
Controlled by arterial pressure	Not stated	Yes	Yes, in "attacks"	Yes, when present	Yes, when present	Same as myalgia
Occurrence at night	Not mentioned	Usually worse at night than day	About 50%	Occasionally	Often	Same as myalgia

TABLE 1.—(*continued*)

CONDITIONS	SLUDER'S SYNDROME	VASCULAR HEADACHE, HISTAMINIC CEPHALGIA	MYALGIA OF HEAD	VASOMOTOR RHINITIS	HYPERPLASTIC SINUSITIS*	MENIERE'S SYNDROME
Reproduced by histamine	Never tried	Yes	Yes	Yes	The vasomotor component	Same as myalgia
Arterial tenderness	Not tried	Yes	Yes	Occasional	Occasional	Infrequent
Palpation reference	Not noted	None	In height of attack	When myalgia is present	When myalgia is present	Infrequent
Effect of vasodilators (cholinergic)	Not noted	Relieves	Relieves attack	Relieves paroxysm	Relieves vasomotor rhinitis component	Infrequent
SYMPTOMS REFERABLE TO EAR						
Tubotympanitis	Frequent	Not reported	Occasional	Frequent	Frequent	Occasional
Mild vertigo on change of position	Frequent	Not reported	Frequent	Occasional	Common	Frequent
Severe vertigo (Ménière's)	Occasional	Occasional (origin of treatment for Ménière's)	Occasional	Occasional	Occasional	Characteristic

TABLE 1.—(*continued*)

CONDITIONS	SLUDER'S SYNDROME	VASCULAR HEADACHE, HISTAMINIC CEPHALGIA	MYALGIA OF HEAD	VASOMOTOR RHINITIS	HYPERPLASTIC SINUSITIS*	MENIERE'S SYNDROME
Tinnitus	Frequent	Occasional	Frequent	Uncommon	Occasional	Characteristic
Deafness	Not reported	Occasional (labyrinthine type)	Occasional (labyrinthine type)	Occasional (tubal occlusion)	Occasional (tubal occlusion)	Characteristic
SYMPTOMS REFERABLE TO THE EYE						
Conjuctival injection	Present	Present, usually unilateral	Frequent, unilateral or bilateral	Usually bilateral	Usually bilateral	Occasional
Lacrimation	Frequent	Present and usually unilateral	Occasional, unilateral or bilateral	Frequent, usually bilateral	Frequent, usually bilateral	Occasional
Edema of eyelids	Occasional	Common	Occasional	Frequent	Occasional	Occasional
Dilatation or narrowing of pupils	Occasional	Occasional	Occasional	Occasional	Occasional	Occasional
SYMPTOMS REFERABLE TO THE NOSE						
Congestion	Present	Usually unilateral	Frequent; unilateral or bilateral	Invariable	Invariable	Occasional

TABLE 1.—(*continued*)

CONDITIONS	SLUDER'S SYNDROME	VASCULAR HEADACHE, HISTAMINIC CEPHALGIA	MYALGIA OF HEAD	VASOMOTOR RHINITIS	HYPERPLASTIC SINUSITIS*	MENIERE'S SYNDROME
Polypoid hypertrophy	Present	Absent	Absent	Frequent	Invariable; usually bilateral	None
Thin discharge	Present	Present, usually unilateral	Occasional; unilateral or bilateral	Invariable	Common	Occasional; usually unilateral and on side of affected ear
Itching and burning of skin	Present	Occasional	Occasional	Frequent	Occasional	Not noted
			SYMPTOMS REFERABLE TO MOUTH AND THROAT			
Glossodynia	Present	Not described	Occasional	Occasional	Occasional	None
Pain referred to ear	Present	Present	Present; superior constrictors involved	Rare	Rare	None
Itching of palate, tonsillar pillar	Present	Not noted	Rare	Common	Common	Not noted
Globus	Present	Not noted	Not unusual	Occasional	Occasional	Not described

TABLE 1.—(*continued*)

CONDITIONS	SLUDER'S SYNDROME	VASCULAR HEADACHE, HISTAMINIC CEPHALGIA	MYALGIA OF HEAD	VASOMOTOR RHINITIS	HYPERPLASTIC SINUSITIS*	MENIERE'S SYNDROME
Hoarseness	Present	Not noted	Arytenoideus involved	Occasional	Rare	None
Pain on swallowing	Present	Not noted	Superior constrictors involved	Rare	Rare	None

*Hyperplastic sinusitis is perennial vasomotor rhinitis with secondary sinusitis.

TABLE 2

DIAGNOSES MADE IN 362 CASES OF INTRINSIC ALLERGY SYNDROME

DIAGNOSIS	CASES
Perennial vasomotor rhinitis only	173
Vasomotor rhinitis* and myalgia	30
Vasomotor rhinitis,* myalgia and vasodilating pain	13
Vasomotor rhinitis* and vasodilating pain	13
Vasomotor rhinitis* and Ménière's syndrome	11
Vasomotor rhinitis,* Ménière's syndrome and myalgia	8
Vasomotor rhinitis,* myalgia, vasodilating pain and Ménière's syndrome	2
Myalgia, Ménière's syndrome and vasodilating pain	23
Myalgia	36
Myalgia and Ménière's syndrome	16
Myalgia and vasodilating pain	5
Vasodilating pain	10
Ménière's syndrome	22
Total	362

*In 75 cases vasomotor rhinitis was associated with other disorders.

TABLE 3

RESULTS OF ATKINSON TEST AND TREATMENT WITH HISTAMINE AND NICOTINIC ACID AMIDE

DIAGNOSIS	RESULTS OF TEST	RESULTS OF TREATMENT NICOTINIC ACID USED IN 49 CASES GOOD*	FAIR*	FAILURE
Ménière's syndrome	Positive	8	..	1
	Negative	4	..	..
Myalgia and Ménière's syndrome	Positive	1	..	..
	Negative	3	..	..
Vasomotor rhinitis and Ménière's syndrome	Negative	1	..	..
Vasomotor rhinitis	Positive	9	3	2
	Negative	6	..	1
Myalgia of head	Positive	2	..	1
	Negative	1	..	3
Myalgia and vasomotor rhinitis	Positive	2	..	..
	Negative	1	..	..
		HISTAMINE USED IN 6 CASES		
Vasomotor rhinitis	Positive		Fair	
	Negative		Very good	
	Negative		Good	
	Strongly positive		Very Good	
Myalgia	Strongly positive		Failed	
Vasodilating pain	Negative		Failed	

*A good result was considered one in which complete or nearly complete relief of symptoms was maintained for 3 months or more. A fair result was considered one in which 50 to 90 per cent relief was maintained for 3 months or more.

TABLE 4

RESULTS OF INTRACUTANEOUS TEST AND TREATMENT WITH HISTAMINE IN 55 CASES

DIAGNOSIS	TOTAL CASES	HISTAMINE SKIN TEST	RESULTS OF TREATMENT GOOD	FAIR	FAILURE
Vasomotor rhinitis	28	Positive	5	1	10*
		Negative	6	..	6†
Ménière's syndrome	1‡	Positive	..	..	1
Myalgia	14	Positive	5	1	3
		Negative	4	..	1
Myalgia and vasomotor rhinitis	4	Positive	3	..	1
Vasomotor rhinitis and vasodilating pain	7	Positive	3	1	2
		Negative	1	1	..
Vasodilating pain	1	Positive	..	1	..

*Subsequent relief with niacin in 1 case and with simultaneous use of histamine and nicotinic acid in 1 case.

†Two patients had asthma. Subsequent relief with niacin in 2 cases.

‡Also positive Atkinson test.

TABLE 5

RESULTS OF TREATMENT WITH HISTAMINE OR NIACIN IN 327 CASES

CONDITION	HISTAMINE			NIACIN		
	GOOD	FAIR	FAILURES	GOOD	FAIR	FAILURES
Perennial vasomotor rhinitis	33 (55%)	7 (14%)	18 (31%)	54 (57%)	10 (11%)	30 (31%)
Perennial vasomotor rhinitis and Ménière's syndrome	0	—	2	8	—	1
Perennial vasomotor rhinitis, vasodilating pain, Ménière's syndrome and myalgia	1*	—	—	3	—	—
Vasomotor rhinitis, Ménière's syndrome and myalgia	0	—	2	8	—	1
Ménière's syndrome	—	—	—	16	3	1
Ménière's syndrome and myalgia	2	—	2	10	3	2
Vasodilating pain syndrome†	2	—	3	3	—	3
Vasodilating pain syndrome and perennial vasomotor rhinitis	4	—	3	6	2	1
Vasodilating pain and myalgia	1	—	2	2	—	1
Vasodilating pain, myalgia and perennial vasomotor rhinitis	6	—	3	2	1	2
Myalgia and perennial vasomotor rhinitis	9	—	3	16	—	2
Myalgia	9	1	4	13	—	6

*Treatment was changed to niacin by mouth after six months with continued relief of symptoms.

†Histamine was given intravenously in this group of cases.

XXXIX

SUMMARY OF SOME KNOWN FACTS CONCERNING THE COMMON COLD

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It seemed to me that it would be of interest and value to summarize all the known facts of the common cold. I scarcely realized how much is actually known. Nor did I realize how difficult it would be to evaluate some of the published items as to their being established facts, or mere probabilities. A better title for this paper would have been, "A Brief Discussion on the Common Cold."

For the purposes of this paper, our meaning of the common cold is that widespread, mild, afebrile, epidemic rhinitis occurring principally during autumn and spring months. It is characterized by copious, thin, mucinous, nasal discharge and a self-limited course of two or three days, which is often followed by or complicated by suppurative infections of the nose, sinuses, ears and lower air passages. There are undoubtedly a number of conditions very similar to the common cold. These may be difficult to differentiate from it at times, but that does not invalidate our conception of the cold as a disease entity with a specific etiology.

It is now well established that the etiological factor is a filterable virus. Kruse[1] determined this in 1914. It was corroborated by Foster[2] in 1916 and again by the masterly work of Dochez and his group[3, 4] in the 1920's. It seems to me that the work of this group is a model piece of careful research and deserves special mention.

They first determined the nature of the normal bacterial flora in the nasopharynx in a group of individuals over a period of months. Then they looked for essential changes in this flora during attacks of colds and found none. They sought for a laboratory animal that was susceptible to colds and found it in the chimpanzee. They studied the symptoms of spontaneous colds in these apes and determined that they were similar to the disease in man. Then, isolating a colony of chimpanzees and exposing them to a patient with a cold, they proved

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that these apes were susceptible to human colds. Again quarantining a colony of apes and keeping them under strict surgical asepsis, they produced colds with the filtered nasopharyngeal washings from patients with colds. They ruled out the tiny-filter passing gram-negative organisms, so frequently found, as the cause by demonstrating that colds were produced when these were absent, that filtrates from normal individuals containing these gram-negative filter passing organisms failed to produce colds, and that fresh cultures of this organism failed to produce colds.

They cultured and subcultured the virus and found that it was still active, after the eighty-eighth subculture. They transmitted colds from one ape to another by means of the virus. They then studied the virus itself and learned the following facts.

It grows on chick embryo medium under anaerobic conditions, but not under aerobic. It becomes inactivated at 56° after a short exposure. It retains its activity for two weeks at icebox temperature and for four months if frozen and desiccated. When passed from one individual to another, it retains but does not enhance its virulence. A culture loses its activity rapidly if removed from the incubator and placed on ice. Acacia in the culture prevents this loss. They differentiated it from the virus of herpes by means of intracerebral inoculations in the rabbit.

They further determined[4] that this virus promoted the growth of pathogenic organisms in the throats of patients with colds.

Olitsky and McCartney[5] found that virus actively transmitted colds if removed during the first 20 hours of symptoms. There is some evidence that contagiousness begins even a few hours before the onset of symptoms.[6] The incubation period was found to be between 8 and 48 hours.

Chapman and Hyde[7] discovered antigenically that the cold virus was different from that of influenza. Shibley, et al.[4] demonstrated on their apes that the cold virus does not protect against influenza.

The normal bacterial flora has been studied by many workers in various parts of the world. It is essentially the same everywhere and contains alpha and gamma streptococci, Neisseria catarrhalis, diphtheroids, staphylococcus albus and aureus, beta hemolytic streptococcus, pneumococcus and Hemophilus influenza.[8] Burky and Smillie[9] and their co-workers who studied isolated communities found the flora to be essentially the same in Labrador, Alabama, and the Virgin Islands. The flora at Spitzbergen also was found by Paul and Freese[10] to be very similar excepting that pneumococcus and hemolytic strep-

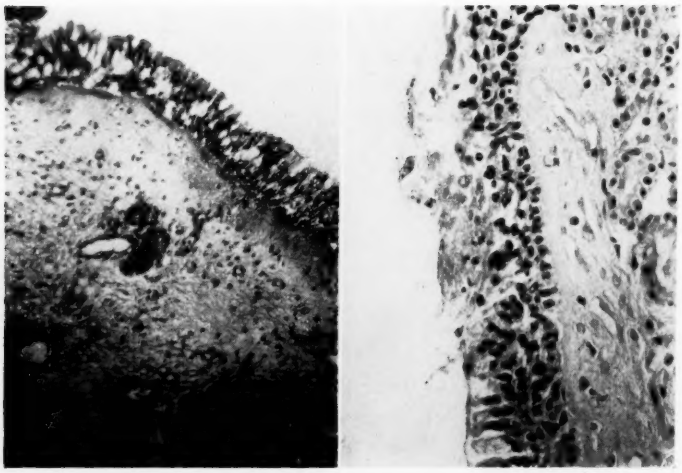

Fig. 1.—Edema of submucosa in early stages of the cold. Specimen removed two hours after onset of symptoms.

Fig. 2.—Necrosis at surface of epithelium on third day.

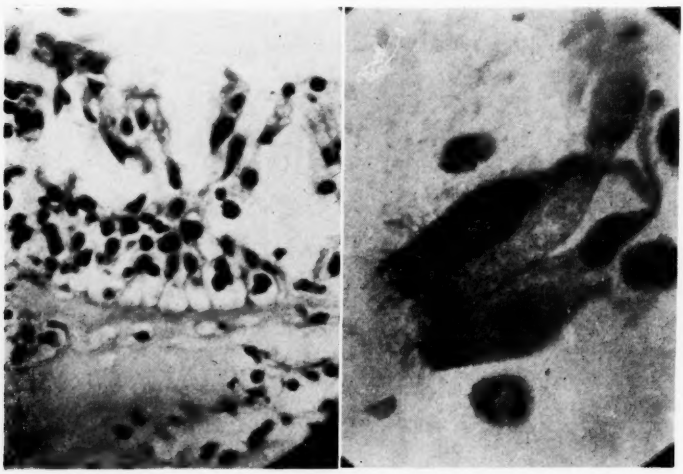

Fig. 3.—Vacuolization of epithelial cells on second day of cold. Some of the loosened cells appear foamy.

Fig. 4.—Group of cells removed from the nasal mucosa by scraping gently on the second day. Two of the cells appear to be secreting mucus. The nucleus of one of these stains poorly as though the cell were necrotic.

tococcus were rare. At some seasons and during some years, one type of organism will predominate above others. These bacteria play a big role in colds, not as a primary cause, but as secondary invaders. They do not seem to have the power to initiate infection. It is these organisms which produce suppurative rhinitis, sinusitis, otitis media, bronchitis and pneumonia. They may be carried by a large portion of the population in a community with impunity until an epidemic of colds occurs, and then they invade, giving to the epidemic its characteristic complications. For instance, Smillie,[11] in 1940, reported a study from an orphanage. Pneumococcus type 14, was present in all of the children, but all were well. Then an epidemic of colds went through the institution. The numbers of pneumococcus, type 14, increased in the throats of the children. Several developed otitis media and two contracted pneumonia. Pneumococcus, type 14, was recovered from the sputum of these two and from the ear discharges of those having otitis media.

Just what the relation between the cold virus and these pathogenic bacteria may be is not clear. Smillie in the study just quoted, Shibley, Mills and Dochez[4] and others have found that the growth of pathogenic organisms is stimulated or promoted by the virus. Whether this is due to direct stimulation of the bacteria, to opening up of better growth conditions, or to reduction of tissue defense does not seem to have been determined.

It has long been held that exposure to sudden temperature changes, especially if it involves chilling and wetting, causes colds. This is such a universal experience that it cannot be disregarded. There have been several controlled observations which seem to indicate definitely that exposure is at least a factor in causation of colds. Shade[12] reported a four-fold increase in incidence of respiratory infection among 17,000 German troops when the weather became severe. Gahwyler[13] made controlled observations on two different occasions. The first involved 2,700 men sent into the wet trenches compared with 5,300 kept in the barracks and the second 4,500 men kept out in a sharp wind for three days compared with 3,500 kept in their quarters. In both instances the incidence of colds was about four times as great in the exposed group as in the protected one. In both instances, apparently there was chilling from wind or dampness. Subjection to dry subzero weather does not seem to have the effect of promoting colds. In fact, in my home community we are apt to be rather free of colds in subzero weather. This fact has often been noted in connection with arctic explorers and other outdoor people subjected to very cold weather.

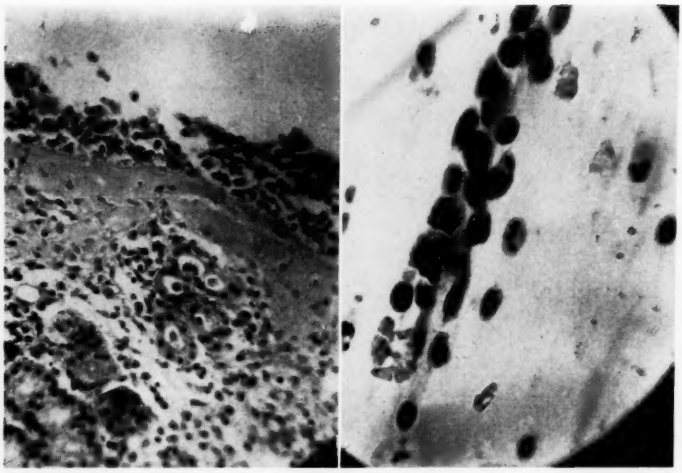

Fig. 5.—Disintegration of the epithelium on the third day. It is possible that this appearance might be artifact.

Fig. 6.—Secretion from the third day of a cold containing many replacement or stellate epithelial cells from deep in the mucosa.

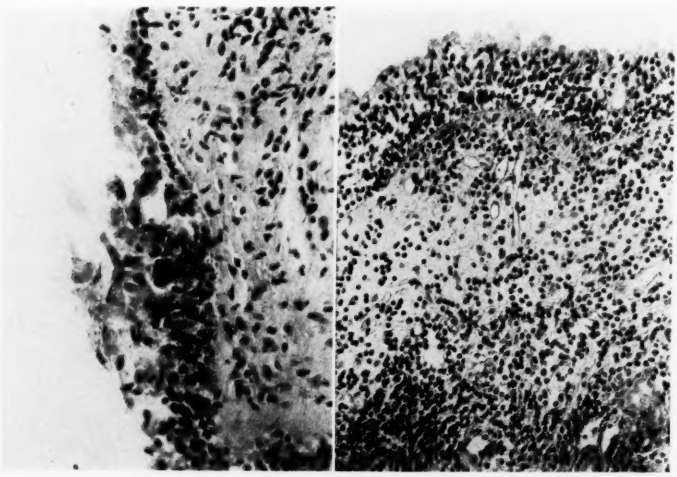

Fig. 7.—Specimen removed on fifth day. In one small spot only one layer of cells remains upon the basement membrane. Here also the question of mechanical damage must be considered.

Fig. 8.—Epithelium on the tenth day of a cold—stage of convalescence. The epithelium appears to be regenerating.

Mudd, Grant and Goldman[14] measured temperature changes in the nose and throat following chilling of distant portions of the body. Under such circumstances, the temperature drop of the nasal mucosa was as much as 6° C.

Spiesman[15] reports a similar study on 63 individuals in which he was able to divide his subjects into three groups according to the promptness, character and duration of the temperature change in the nose. The first group was normal and manifested a prompt drop in temperature of the nasal mucosa with a return to normal in about ten minutes. The second group was composed largely of individuals who frequently suffered from colds. In this group, both the temperature drop and the return to normal were delayed. Then there was a third, or hypersensitive, group in which there was a rise instead of a fall.

These studies would seem to supply experimental basis for the relation of colds to chilling.

In summing up the etiological factors it appears to be reasonably certain that the cause of colds is a specific filterable virus, that bacteria are secondary invaders and that chilling is a prominent predisposing factor and acts by reducing the temperature in the nasal mucosa.

The cold is a widespread communicable disease. According to Smiley[16], the incidence in the U. S. Navy is about 2,500 per thousand per year. He submits evidence[17] that most colds occur in cold susceptible groups. Keefer[6] reports 2-3 colds per year per person among civilians. Greenspan[18] reviewed ten surveys and reported the figures as varying between 277 to almost 4,300 per thousand per year. Coryza is about equally prevalent everywhere. Mild climates seem to have no advantage over the severe ones in this respect.

Reports from institutions, families and isolated communities as well as the experimental studies reported, establish the fact of the contagiousness of colds.[3, 10, 11] Smillie reported outbreaks on St. John's Island, an isolated community in Alabama and in Laborador with a single source of infection in each case. In each case, he observed and recorded the spread through the community in question.

The spread could be controlled, theoretically by isolation, since the spread is largely by droplet infection. But people will not submit to isolation. For some strange reason, cold sufferers consider themselves as self-sacrificing good sports when they remain at work rather than at home.

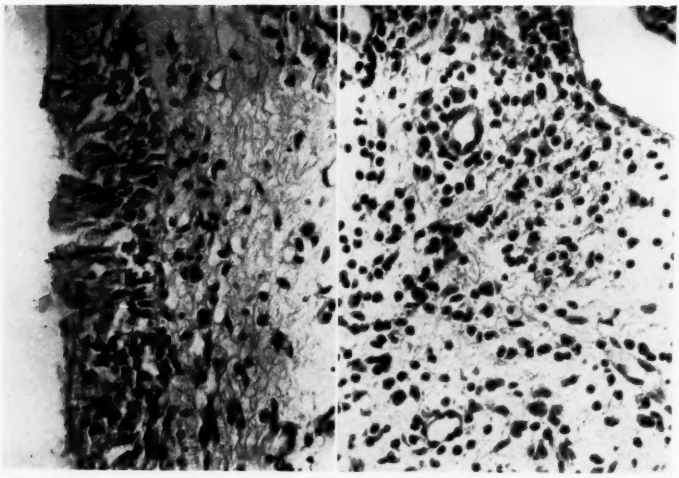

Fig. 9.—Epithelium on the fourteenth day. Imperfect columnar cells are present. They might be in the process of regeneration. On the other hand, such imperfect cells are found in normal epithelium in the zone of transition from squamous to ciliated cells in the fore part of the nose.

Fig. 10.—Cellular infiltration on the third day.

There are three peaks in the incidence each year in most communities. They are September-October, January-February, and April-May. It is my impression that the winter peak in our community comes considerably later than January-February. That is the period of our dry, cold weather when we are reasonably free of colds. The incubation period according to Keefer[6] is 24 to 36 hours and he offers evidence that the patient is most infectious during the first day and may be capable of spreading the disease as much as four or five hours before his symptoms begin.

The pathological picture seen in the nose during colds is that of an inflammatory reaction of the mucous membrane. It is swollen, red and covered with profuse discharge. The air passages are frequently occluded by swollen membrane, secretion, or both.

Fifteen years ago, we took 25 specimens for biopsy from the inferior turbinates of cold sufferers during various stages of the disease and about 100 scrapings from the epithelium.[19] From these we attempted to reconstruct the steps of the pathological changes in the

mucous membrane. It was not easy to differentiate between changes caused by disease and those caused by crushing action of the instruments which removed the specimens. Judging as carefully as possible, we reported the steps to be about as follows. First, there develops an edema of the submucosa, followed by an infiltration of macrophages and a few granulocytes. The first change to appear in the epithelium is a separation of the cells as though a fluid formed between them. They loosen and the surface cells begin to slough off. Some become vacuolated or foamy in appearance. A necrotic zone may form next to the surface or single cells or groups of cells may show necrosis. This necrosis may extend deep. Then almost the entire epithelium disintegrates; it looks as though it had been exploded. Many and sometimes all of the columnar cells are gone by the third day. Some of the stellate cells close to the basement membrane also slough out. During this stage, the nasal secretion may contain many epithelial cells, many of which are surmounted by vigorously active cilia. Sometimes there may be only a single layer of cells left on the basement membrane. The cells that remain proliferate after the most acute stage has passed until they are many layers deep. A syncytium forms at the surface. Finally the surface cells begin to lengthen until they again look like columnar cells. The cellular infiltration of the submucosa is largely composed of variously shaped mononuclear cells with a scattering of polymorphonuclears and some eosinophiles. The edema soon decreases, but the cellular infiltrate increases. By the second or third day, many granulocytes and macrophages are found in the epithelium on their way to the surface. They increase in number as the purulent stage develops.

Repairs seems to take place even while there is still much secretion in the nose. A many layered epithelium full of granulocytes was found on the eighth day, and, on the tenth day, the surface cells showed elongation and on the fourteenth day, definite columnar form.

No bacteria were ever found in the epithelium or submucosa, only in the secretion. Neither were any inclusion bodies ever found.

That is the way it looked 15 years ago. I still believe that this picture is essentially correct. The changes in the submucosa were definite and there was not much chance for error. There is no doubt that the surface cells slough out of the epithelium because large numbers of living ciliated cells are found floating in the secretion and on the second and third days going down as it were with all cilia firing. I have watched ciliated cells under the microscope shake themselves loose from the epithelium especially under stimulation of a slightly

alkaline pH and actually have a movie of one such cell. Cells from deep in the epithelium are also found in large numbers in the secretion.

There is still the possibility, however, that the steps which we observed in the biopsy specimens were produced or at least modified by the crushing action of the instrument used for excision. It is so easy to knock off the cells and so difficult to avoid doing so. We tried to confine our study to areas which had been protected by folds, or which were overlaid by the secretion or necrotic cells that looked as though they were produced where they lay. Mechanical damage was excluded as far as possible.

Those areas which showed newly developing columnar cells are open to doubt. When the surface cells have been lost and the deeper cells grow out, elongate and become ciliated, they must of necessity pass through such a transitional stage. Likewise, however, there is in the normal epithelium a transitional zone between the stratified areas in the fore part of the nasal cavity and the ciliated columnar further back. These zones are inconstant in position. It is possible that the biopsy of the specimen taken on the fourteenth day of a cold which showed what we took to be newly developing cells, might have been taken from a transitional zone. Perhaps that area had always looked the way we found it to be.

There is doubt also as to what constitutes necrosis from the action of toxins in cells. Cells which have just secreted or are about to secrete mucus look necrotic. Physiologists have told me that it is not known if a secreting epithelial cell survives to secrete again.

Since this study was made 15 years ago, no one seems to have done anything similar to it. It should be repeated by someone else. Shope found very similar changes in the bronchial epithelium of swine afflicted with swine influenza. He described fragmentation of the epithelium and loss of surface cells and published photographs which were strikingly similar to the appearances in these specimens just described.

The outstanding clinical feature of a cold is the profuse, thin secretion. The physicial differences between this and the normal secretion are obvious. The chemical differences have not been well worked out.

I made some attempts to study the lysozyme content about ten years ago,[20] and in seven cases studied found that the lytic power of the secretion was reduced at the onset of symptoms (and in one

case several hours before) and increased again just before improvement began. It seemed quite definite that this was not a dilution phenomenon because the lytic power was found low before there was much of any secretion and high when improvement had begun even in the presence of copious secretion. This study was simply a preliminary one on a small group. Cahn-Bronner[21] reported similar studies in 1942. He corroborated our findings and went further. He found that the lytic power was not reduced in the copious secretion of hay fever and was therefore convinced that the change in colds is not a dilution phenomenon.

The pH has been tested by a number of observers. Buhrmester[22] found the average pH increased very slightly during colds, that is, the secretion was more alkaline. Fabricant[23] found a very considerable increase and felt that it was of considerable significance. In my tests,[19] it was increased slightly on an average during the acute stage of watery discharge and slightly reduced during the purulent stage. Mittermaier[24] found it reduced during the purulent stage. The slight rise during the acute stage in our series seemed to be too small to be of much significance. My readings were all higher than those taken in situ, probably because of the escape of CO_2. The normal mucus has a range very close to neutrality—7.0.[25] The low readings found by Fabricant[23] have not been corroborated and were apparently due to a fault in technic as pointed out by Nungester.

I have recently made some tests to determine if an increase in pH (alkalinity) is in itself irritating or destructive in the nose as suggested by Fabricant. The material consisted of a series of bottles containing physiologic saline each with a different pH. The range used was from 6.8 up to 8.4. This saline was dropped into the noses of a number of volunteers in comparatively large quantities (half a dropper full or more) at frequent intervals. Up to pH 8.4 it was found to be so bland as to be practically without sensation. There was no increase of secretion or any other change noted.

Apparently there have been no studies on the pathologic changes in distant parts of the body. The symptoms would indicate that there must be some. For instance, the delayed reaction of the nasal temperature to chilling of the back which was reported by Spiesman;[15] the extreme sensitiveness to any draft experienced by the patient; the sweating and polyuria, which frequently occur, and the salivation, which sometimes occurs, would seem to indicate some disturbance of the sympathetic-autonomic balance. Spiesman presents some evidence indicating that emotions have a bearing.

There has been a very impressive amount of effort expended upon preventing colds. The results have been disappointing.

Vaccination by bacterial vaccines has been given very thorough trial by the subcutaneous and oral routes and even locally in the mucous membrane. Reports have been both favorable,[26-29] and unfavorable.[30-34] One author who presents strikingly good results from subcutaneous vaccination writes that, "No control series was used, as the primary purpose of the inoculations was to do something for the students and not to test the vaccine. It did not seem fair to exclude students wishing vaccination simply to provide a control group for statistics." Another author, using the same method, reduced the incidence of colds in a group of students equally successfully—in fact by 55 per cent.[31] However, he vaccinated a control group with normal saline and obtained even more favorable results in this control group, namely 61 per cent reduction.

Vaccination by bacterial vaccines both by mouth and subcutaneously appear at this time to be valueless for reducing either the incidence or severity of colds.

Vaccination by nasal spray[29] has a scientific basis in the fact that immunity may be local. Walsh has demonstrated that the antibody titer of the mucosa can be raised as compared with that of the blood and other tissues by means of local application of bacterial vaccines. His results look promising as far as complicating bacteria are concerned, but are not aimed at the virus itself. This work should be pursued further and corroborated by others.

Unfortunately, both the cold virus and the group of pathogenic organisms usually found in the nasopharyngeal flora are very weak producers of antigens. One would scarcely expect any notable results from vaccination. The immunity following an attack of coryza lasts only three or four weeks. Likewise, infections by staphylococci and streptococci in general produce no lasting immunity in patients who recover.

These studies in large groups of students or industrial workers are severely handicapped by the human element and must be controlled with exceedingly great care. This fact is demonstrated by the control groups which were so strikingly benefited by injections of saline. Students who come requesting vaccination, even before the study is begun, are already a selected group in whom the results are probably heavily weighted by the will to believe.

Dochez[3] tried vaccination with heat killed virus by hypodermic injection. The results seemed promising in apes but were negative

in man. He suggests that the reason might lie in the supposition that immunity is not general, but may be local in the nose. This virus vaccine should be tried in a nasal spray. Unlike the cold virus, the influenza virus does produce an antigen and reports on results from vaccination appear to be favorable.[35]

Vitamins have been tried in large groups of patients and have been found to be without value in preventing colds.[36] Crampton[37] reports favorably upon the use of vitamins but offers no control series.

The sulphonamides have been tried and most reports indicate negative results as far as the cold itself is concerned. On the other hand, they have very definite value in the prevention[8, 38] and treatment of complications. That does not mean that they should be used routinely.

McCord[39] and McConnell[40] both made studies of the relation of air conditioning (as it exists now in office buildings) and the incidence of coryza. Each recorded the number of colds among employees working in an air-conditioned building as compared with a similar group working in an old building in which there was no air conditioning. In both studies, the incidence was the same in both groups. Both of these studies are open to question, because the employees remained under the study conditions only eight hours in each twenty-four.

There is another type of air conditioning which is aimed at destruction of bacteria and viruses floating in the atmosphere which seems to be very effective in stopping the spread of infection. The purification can be accomplished either by aerosols,[41] antiseptic chemicals, or by ultra-violet light.[42] The latter seems to be very promising. It can be used either by subjecting the whole room to ultra-violet rays, or just the source of air, or it can be used in the form of barriers of light. Wells states that the foundations for efficiently purifying air and checking its purity are as well established as the control of the purity of the supply of drinking water. He believes that such control and purification of air supply is necessary to free us from air-borne infection. He likens the contamination of air supply by the nasopharynx to the contamination of water supply by the excreta from the intestinal tract. Perhaps in this we have an effective method of preventing colds in schools and factories.

The hardening process has long been advocated as a cold preventative; that is, cold baths, outdoor exercise and calisthenics. It may have value, but has not been subjected to enough well controlled

studies to have established a value.[18] Outdoor individuals do have fewer colds, but they are also subjected to fewer contacts with infection.

In general, treatment has been as disappointing as prevention and it is just as difficult to determine the value of the suggested remedies. Colds are self-limited unless complicated. Some are abortive and are over in a few hours. Some last only 24 or 48 hours. And others, when complicated, run a course covering several weeks. When symptoms disappear after the exhibition of some remedy, it is difficult to know if the remedy exerted some beneficial influence, or if the attack was about to subside anyway.

Codeine and papaverine[43] seem to have some definite value in shortening the course. Under some circumstances this remedy should doubtless be used.

Steaming often gives symptomatic relief.

If serious complications threaten, then the sulfonamides are indicated.

Isolation should be practiced whenever feasible. It not only prevents spread of the cold virus, but protects the sufferer from bacterial infection of his damaged epithelium by droplets from other individuals. It seems to me that this is very important. If bacterial infection can be prevented, the attack will soon be over because the virus stage is so short.

Of all of the many nasal drops which have been advocated, simple ephedrine in saline solution seems to give about as much relief from nasal obstruction as anything else. This is of course purely symptomatic treatment, and I fear is not resting upon a firm scientific basis. Why do we want to open the nasal passages when nature has closed them? Perhaps the closure is purposeful. In my practice, I have done the opposite. As soon as there is any sign or symptom of an approaching cold, I have instructed my patients to block the nostril (or nostrils) with clean cotton, tightly enough to reduce materially the inflow of air. The cotton must be changed frequently. It seems to me that the irritation is greatly reduced by this simple measure and often the swelling does not become great enough to obstruct the air passage.

Much treatment has been directed toward the cilia, generally with a view toward stimulation. But do we know if the cilia need stimulation during a cold? Perhaps they are working at capacity as

it is. In some cases, they are largely destroyed. Under such circumstances stimulation would seem to be irrational.

If stimulation is desired, it can be had by means of a mildly alkaline solution (8.0—8.5) which contains magnesium and calcium salts.[44] At pH 8.5 in Tyrode's solution, the cilia will continue active for 24 hours. Acids, on the other hand, paralyze ciliary action. It ceases entirely if the pH drops to 6.4 or less. If magnesium or calcium ions are omitted, the tissue breaks up and action ceases. The sodium ion according to Negus is the strongest inhibitor of ciliary action.

CONCLUSIONS

These facts about the cold seem to be fairly well established. It is a widespread, contagious epidemic disease which occurs in all climates, especially in the autumn and spring months. It is spread by droplet infection and can be controlled by isolation like other communicable diseases. It is caused by a specific virus and is frequently followed immediately by complications due to bacterial infection. These are purulent rhinitis, otitis media, laryngitis, bronchitis. Chilling and exposure are definite etiologic factors probably because of prolonged reduced temperature in the nasal mucosa. Immunity following attacks is very brief both as far as the virus and the complicating bacteria are concerned. Vaccination is unsuccessful, apparently because both the virus and the bacteria have such poor antigenic properties.

Prevention by the giving of vitamins, or by so-called "hardening processes" are also largely unsuccessful.

The local pathology centers about injury to the epithelium, presumably by direct action of the toxins on the epithelial cells, and consists in destruction of the superficial cells. There is a marked submucosal cellular reaction. The secretion increases tremendously in volume and contains a lower percentage of mucin. There is at least some indication that the lysozyme content decreases even before the volume increases. The change in pH is small, if any, and tends toward the alkaline side.

Systemic pathological changes are largely unknown.

Treatment is not very successful and consists principally in symptomatic measures and attempts to prevent complications.

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XL

SOME PROBLEMS IN THE SURGICAL TREATMENT OF BILATERAL ABDUCTOR PARALYSIS OF THE LARYNX

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The object of this paper is to acquaint those laryngologists who have not had experience in the surgical treatment of bilateral abductor paralysis of the larynx with some of the problems I have met in the handling of these cases by arytenoidectomy, and to give some advice as to the best place to make the incision. If the case is one where the patient has not had a tracheotomy, it is very likely that the operator will feel that a straight line incision, from the hyoid bone to the sternum, will be the incision of choice because it is natural to believe that you will be able to get a complete exposure of the trachea and the thyroid cartilage and thus obviate the necessity for a horizontal incision in addition to the tracheotomy incision, which so far has been a part of an arytenoidectomy.

If you have a case in which one thyroid operation has been performed by an excellent operator, where the pretracheal muscles have not been severed and where there was primary healing without distortion and the formation of scar tissue, it is probable that you will have no trouble in exposing the muscles covering the thyroid cartilage to your satisfaction. However, you must now completely expose the right wing of the thyroid cartilage and reflect upward the thyrohyoid muscle to expose that part of the thyroid cartilage through which you are to make the window for the extraction of the arytenoid. This is sometimes most difficult to accomplish under the best conditions without using a horizontal incision. When using the straight midline incision you must exert an unusual amount of traction upon the pretracheal tissue to expose the thyroid cartilage properly or you must displace the larynx in such a way that it will be a long and difficult task to locate and remove the arytenoid cartilage. I would not advise the midline incision for the beginner.

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If, however, a patient comes to you for relief of his paralysis and he has had two or three thyroid operations together with x-ray treatment, the probability is that there is nothing left of the pretracheal muscles but a mass of scar tissue which is adherent to all the surrounding structures, and it may be barely possible to find a line of cleavage along which you can cut, even when using the horizontal incision. It would, therefore, be impossible to separate and retract this tissue if you were using a midline incision. I feel, therefore, that the easiest and proper incision for the expeditious performance of the arytenoid operation is the horizontal incision.

In short, fat necks with a small thyroid cartilage and trachea, you may find some difficulty occasionally in locating the lower border of the thyroid cartilage. It was a surprise to me when I first had the experience of spending some time dissecting the cricoid cartilage free of scar tissue, thinking it was the thyroid cartilage. This disorientation was brought about by the fact that I was operating upon a short, thick neck, full of scar tissue, where only with the most careful and tedious dissection was I able to expose the upper part of the trachea. This mistake on my part was due to the fact that I had made my incision at the lower border of the cricoid cartilage instead of the lower border of the thyroid cartilage. When I have a difficult case now, I make the horizontal incision longer and dissect the skin and subcutaneous tissues well upward and push my index finger over the trachea and up over the thyroid cartilage until I orient myself by feeling the thyroid notch. This accident may not happen to you, but it may shorten the time of the operation if you will use the patience necessary to insure the proper position for the horizontal incision.

I might further add that the majority of the 34 patients which I personally have operated upon have had two or more thyroid operations and necks filled with scar tissue, so that most careful dissection was necessary in some of them in order to avoid the vessels within the carotid sheath. In some of these cases the scar tissue will form about the carotid sheath and pull it from under the protecting margin of the sternocleidomastoid muscle. When this happens, care must be exercised not to cut into, or to put the point of a pair of sharp dissecting scissors through, the jugular vein. Sometimes the thyroid cartilage itself will be so covered wih scar and muscle tissue that it is difficult to expose satisfactorily the area in which you want to make your window. The posterior border of the thyroid cartilage and the attachment of the inferior constrictor muscle may be so thickened and fixed in scar tissue that a satisfactory digital examination for the

location of the arytenoid cartilage cannot be made. If circumstances and the case warrant it, a submucous dissection of this area may be performed. So far I have tried this in only one case.

Occasionally the arytenoid cartilage will be very elusive, especially in young people. The tissues are soft and pliable; the arytenoid cartilage is freely movable and may be placed rather deep in a broad larynx.

Under such circumstances the position of the arytenoid cartilage and the facet of the cricoid cartilage may be readily located by placing the index finger of the left hand behind the posterior border of the thyroid cartilage and feeling the upper border of the cricoid cartilage through the fibers of the inferior constrictor muscle. If the point of the dissecting scissors is then pushed through the fibers of the arytenoid muscles which are exposed after making the window, you will be able to locate the position of the arytenoid cartilage and feel it move between the point of the scissors and the tip of your finger. By this means it is easier to find the location of the cricoarytenoid articulation and sever the capsule. In a like manner, the fibers of the arytenoid muscles may be separated and cut to expose the outer surface of the arytenoid cartilage. From there on it is a matter of patience and judgment until the arytenoid cartilage is delivered to your satisfaction.

In making the window through the thyroid cartilage, care should be exercised not to make the window too large. In our earlier cases we were content in placing the suture fixing the cord away from the middle line through the external perichondrium of the thyroid cartilage, but now we are fixing this suture by putting it through the cartilage and the perichondrium at the upper part of the anterior border of the window. If the window is too large, the suture may be placed too far forward on the cord to give a satisfactory voice.

The problem which made us change to putting the fixing suture through the thyroid cartilage was the severe paroxysms of coughing which sometimes follow the introduction of a tracheotomy tube into the larynx of a patient who has never before worn one. Occasionally it is very difficult to control this coughing and I believe that in one of our recent cases this coughing caused the breaking away of the suture which had been placed through the external perichondrium alone. Because of this coughing complication, we have recently had to remove the tracheotomy tube in one case on the second day, and in another on the third day. I know that I have made the statement

that I believed that some of our good results were brought about by keeping the tracheotomy tube in place for as long as four, five or six weeks, but in nervous, excitable patients we must choose between what we would like to do and what is the best thing to do for the individual when an emergency arises. Both of these patients had their tracheotomy tubes corked two hours before the tube was removed. This was done to make sure that there would be no immediate dyspnea. We find that the corking of the tube alone sometimes relieves the coughing.

This complication of postoperative coughing brings up the problem of the best intralaryngeal area in which to place the fixation suture. If this suture is not put securely into the fibers of the thyro-arytenoideus muscle or into the fibrous tissues of the cord itself, it may give way at this point just as well as from the external perichondrium. If the fixing suture is placed too far posteriorly into the muscle fibers of the crico-arytenoideus, it will not be very secure and it will be difficult to get the proper separation of the cords without too much distortion of the tissues in the posterior part of the larynx. In the presence of severe coughing a suture in this area may pull through these muscle fibers. Also, because of its proximity to the inferior constrictor muscle, severe gagging and vomiting may affect its position. It has been our practice to use the laryngoscope before determining the position in which to place the suture. The anesthetist or the assistant uses the scope and the operator with a small, fine hook pulls the cord from the median line of the larynx at several points. The location giving the desired airway with the least amount of separation of the cords anteriorly is the point of choice. This is usually just anterior to the location of the vocal process of the arytenoid cartilage. It might be of interest to note here, that one of the laryngologists watching this operation suggested that we use more than one suture in fixing the cord. I think this is a good suggestion and we have followed it in one case. However, we must be careful not to separate the cords too much if we are anxious to retain a good speaking voice.

After the cord has been fixed, the closure of the laryngeal mucous membrane is the next step if there have been any lacerations of this tissue. The lacerations should be sutured with a fine eye needle, using triple O catgut, and the fibers of the arytenoid muscles should also be pulled together. After suturing the thyrohyoid muscle as nearly as possible to its original attachment to the thyroid cartilage, the wound is ready to close. We still use a drain which is removed anywhere from the second to the fifth day.

The next group of surgical problems to be considered are those which we have encountered in attempting to correct our failures by re-operating upon the same side. I have tried this in three cases and failed. The patient I operated upon the second time on the same side was one I first operated on in November of 1941 and re-operated on in February 1942. At this time I went through the old incision, uncovered the window previously made in the thyroid cartilage, and attempted to put a suture into the location of the cord and further displace it from the median line of the larynx. This was a failure so far as the patient was concerned. However, I felt that I had accomplished something at the time. This patient later developed a granuloma of the larynx which was removed by our bronchoscopic department. I made no further efforts in secondary operations until after I had had a successful result in a case of laryngeal stenosis.[1]

This was the case of a young man 23 years of age who had a laryngeal obstruction with fixation of both arytenoid cartilages. He had been in the hospital some 76 times in the course of three years. He had considerable hypertrophy of the mucous membranes of his larynx and a thickening of his thyroid and arytenoid cartilages. It was thought that removal of one or both of his arytenoids might increase his airway. He was prepared for an arytenoidectomy, and at the time of operation it was found that he had an old perichondritis causing the obstruction. One arytenoid cartilage was removed but on laryngoscopic examination the amount of space gained was negligible. I then decided to remove all the cartilage from one side of his larynx and sew the internal perichondrium and the thyrohyoid muscle to the median surface of the pretracheal muscles, hoping that the formation of scar tissue and the fixation of this side of the larynx to the under surface of the muscles mentioned would widen the larynx laterally. The result was most encouraging. Two months following this operation he had sufficient airway to allow us to close a tracheal fistula which had existed since he was two years old—the result of necrosis of the tracheal rings following a diphtheria infection complicated by a tracheotomy. He was able to get a gainful occupation for the first time in his life.

With this experience to justify my again attempting to correct failures by operating on the same side, I re-operated upon two of the early failures within a short time—one a nurse from the South and the other a housewife from upper New York State. In the case of the nurse, I removed the right wing of the thyroid cartilage and carried out the procedure of suturing the internal perichondrium of the

larynx to the pretracheal tissues, as I had done in the young man. In the housewife, in addition to removing the right wing of the thyroid cartilage, I did a thyrotomy and performed an intratracheal removal of the arytenoid cartilage on the opposite side. Removal of the arytenoid cartilage at this time was done because on laryngoscopic examination at the time of operation, we were unable to widen the larynx by pulling on the sutures put through the internal perichondrium. I thought that the intratracheal removal of the opposite arytenoid cartilage would give me more airway.

The results in these two cases were disappointing. I have not heard from the nurse since three weeks after her operation, which was last summer, and I take it for granted that her result was not satisfactory. However, the lady from upper New York State has been a most grateful and obliging patient, and in her case there was a perceptible narrowing of the larynx after this operation and on indirect examination of the larynx the action of the inferior constrictor and cricopharyngeus muscles could be followed through the right side of her larynx. There was a distinct movement of the right side of the larynx each time she gagged or strained when I was examining her. The arytenoid prominence on the right side, which previous to the second operation remained firm in its position, now drooped toward the interior of the larynx. While both of these cases were between 50 and 65 years of age, which I believe is the unfavorable period for consistent good results, I am nevertheless convinced that we must conserve the integrity of the thyroid cartilage, as far as possible, for the success of any operation to relieve bilateral abductor paralysis of the larynx.

The one point that I did not take into consideration when drawing an analogy between the success that I had with the young man with laryngeal obstruction and the possibility of getting results on these two patients by following the same technique, was that in the young man I was dealing with an obstruction and not with a paralysis of the muscles of the larynx. Within the last three weeks I have done a secondary operation on a young lady who had two previous operations for the relief of her laryngeal paralysis at another institution. The first operation was done about three years ago by the King technique, and the second operation was done about six or eight months later, using the technique of the arytenoidectomy. She came to our clinic about a year ago and we told her that we thought she had sufficient airway to carry on without her tracheotomy tube, but she was reluctant to do this because of the distress that she experienced at night. She was becoming very nervous and felt that she

could not hold a position. We finally persuaded her to let us remove the tracheotomy tube without closing the tracheotomy opening and encouraged her to seek employment, which she did. However, after following our advice for some months, she came to the hospital and said she was ready to have her third operation if I thought it would give her more airway. We did the usual arytenoidectomy on her left side. On exposure of the arytenoid cartilage it was noted that the cartilage was very large and after it was delivered, the left cord fell away from the median line, giving the patient sufficient airway without fixation suture. However, in view of the fact that this patient had been operated upon on two previous occasions for the relief of her paralysis, I was hesitant about not putting in a fixation suture although it gave us more separation of the cords than we deemed necessary. There is no question of the success of this last operation from the standpoint of an airway, and I am hoping she will have a useful voice.

In closing I think there are two more problems which are worth mentioning at this time. One is the question of primary healing. I noticed that in two of our elderly patients the wounds did not heal readily. In one the wound broke down and several bits of cartilage were extruded. The wound healed with an ugly scar although the result was very satisfactory. In this particular case we were longer than usual in doing this operation and several times I could feel the heat of the two lights we were using through my rubber gloves but thought nothing about it. However, when in a second case of an elderly person who should have made an uneventful recovery there was trouble with healing, I began to realize that the heat from the lights we were using was drying out and devitalizing the tissues in these old people and preventing primary repair. I have now changed our procedure and we use one light until the thyroid cartilage is exposed and from there on the operation proceeds with the reflected light of a head mirror. We have had no breaking down of wounds in elderly people since making this change.

The other problem to be considered is that in doing this operation under local anesthesia and injecting the intratracheal tissues through the window made in the thyroid cartilage, care must be taken not to inject too much solution. If the tissues are flooded, it is more difficult to locate and extract the arytenoid cartilage and, in separating the cords, you may have too much separation when the fluid has been absorbed and the tissues return to normal.

These are some of the problems we have met in the surgical treatment of bilateral abductor paralysis of the larynx. If the ex-

periences outlined here prevent the laryngologists who may be enthusiastic enough to undertake this work to avoid some of the embarrassment and pitfalls we have experienced, I will feel that our efforts have not been in vain.

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XLI

ANESTHESIA IN PERORAL ENDOSCOPY

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To the recent revolutionary changes that have taken place in surgical anesthesia, the endoscopist has contributed but little. He has made use of the improved methods of preventing pain that are so essential to good surgery and this may be his best form of repayment to those leaders in anesthesiology who have given him so much.

It is fortunate that anesthesia, which is an integral part of surgery, should have been the subject of serious study and investigation during the past ten years. Out of this study have come new truths and a better understanding of the physiology of respiration, the action of anesthetic agents and the improved methods of their administration.

The endoscopist makes use of all types and methods of anesthesia. The endoscopic examination of the air and food passages is not always possible under local anesthesia despite the instruction of certain teaching institutions of good repute. The seasoned endoscopist with deft technic may, by the judicious use of sedative drugs, be able to carry out most endoscopic procedures with the aid of a local anesthetic. The casual endoscopist whose training may be of the best but who is called on only infrequently to do this work will have in mind his lack of finished technic. He will constantly remember the safety of his patient and for this reason he will, frequently, choose a general anesthetic and so be spared the problem of a struggling, complaining, uncooperative patient. He will look forward to the day when, with added experience, he, too, will be able to carry out endoscopic examinations and treatments under local anesthesia.

Routine methods, following fixed rules, do not make for good anesthesia. Individual cases call for specific types of preanesthetic preparation and a personalized type of anesthesia. The child, the nervous apprehensive adult, the aged, the cachetic, the hypertensive and the patient with disturbance of the respiratory function, all

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these present varying problems that can be met only by the proper choice of anesthesia.

The choice of a preanesthetic agent is predicated on the type of anesthesia to be used and not on the nature of the surgery to be performed. Preanesthetic medication should be individualized and the blind following of a routine in the prescribing of narcotics and hypnotics will bring unnecessary trouble to the anesthetist and the surgeon.

The purpose of the preanesthetic drug is manifold: to allay the fears of the patient, to reduce the amount of anesthetic required, to induce and continue a state of anesthesia, to dry secretions and to counteract the toxic effects of other drugs. Keeping in mind the purpose of the preanesthetic medication, a proper selection can be made for the individual patient whether it be a child or adult.

It is the wish of every surgeon and the hope of the anesthetist that the patient come to the operating room in a state of tranquility. This happy state will be assured if the patient has enjoyed a restful, dreamless sleep the preceding night. Such sleep can be expected, if the patient is not in pain, by the use of a hypnotic of the barbiturate group. Of this group, pentobarbital sodium, more commonly referred to as nembutal, is satisfactory. Nembutal gr. 1½ given with hot milk at nine in the evening will result in a night of sleep and will do much toward preparing the apprehensive patient for what is to many a terrifying ordeal: the trip to the operating room.

Of the preanesthetic agents commonly used, morphine, the most widely used, is the best understood in its action of all the narcotics. It is given not only to control pain but to produce a mild state of narcosis preliminary to the administration of the anesthetic. Morphine is valuable whether the anesthesia is to be of a local or general type. It is usually combined with atropine or scopolamine since the combined medication results in a synergistic effect that augments the physiological action of both drugs.

Atropine acts on the parasympathetic nervous system, chiefly at the periphery, to produce an arrest of the secretions, smooth muscle paralysis and bronchial relaxation. In combination with morphine, it is possible to obtain the same pharmocological effect with a smaller amount than is possible with either of the drugs when used alone. It is for this reason that morphine and atropine or scopolamine are combined to produce the ideal preanesthetic state.

Scopolamine acts like atropine on the peripheral parasympathetic nervous system to arrest secretions and cause smooth muscle paralysis but in addition it has a marked central nervous system effect, probably on the hypothalamus. The combination of scopolamine with morphine has the same helpful effect as does the combining of morphine and atropine. Dogliotti[1] observes that, "The slight hypnotic action of morphine upon the cortical hemispheres, together with the action of scopolamine upon the diencephalon with depression of the motor centers and the association pathways, results in an association of two actions in a particularly favorable manner upon the production of unconsciousness with almost complete absence of excitability and resulting deep sleep." This is almost the ideal state for any endoscopic procedure whether under local or general anesthesia.

In practice, this ideal situation is accomplished by the use of morphine sulfate gr. 1/4 and scopolamine gr. 1/100. If given to an adult one hour before administration of cocaine anesthesia for peroral endoscopy, the patient will be quiet and cooperative and the amount of cocaine necessary to produce satisfactory anesthesia will be reduced to a minimum.

It has been found that combining morphine and scopolamine as a preanesthetic medication is helpful in all types of anesthesia, local with cocaine, general with ether and intravenous with pentothal sodium. There are some side effects that are disagreeable since twenty per cent of patients are nauseated by morphine. A small percentage of people are rendered excitable and a still smaller group develop a pallor and may faint following the administration of morphine. No way is known of anticipating these unpleasant side effects though none of them are of serious import. When morphine and scopolamine are combined the ratio of 25:1 is followed, i. e., morphine gr. 1/4, scopolamine gr. 1/100; morphine gr. 1/6, scopolamine gr. 1/150.

As a preanesthetic agent the barbiturates have been widely used. They are hypnotic and not analgesic in their action, with the maximum effect appearing in about thirty minutes. With the quick-acting types such as seconal and nembutal, the hypnotic effect continues for only a few hours. These barbituric acid drugs are of value in cases where it is planned to use cocaine or novocain as the primary anesthetic since they do much to protect against the convulsive reactions of cocaine and allied drugs.

Endoscopic procedures in children between the ages of four and twelve can sometimes be carried out with no anesthesia if preliminary

use is made of one of the short-acting barbiturates. An opened capsule of seconal placed in the rectum one hour before the examination will often be sufficient to render a child quiet and controllable. The surgeon and the anesthetist will keep in mind that the barbiturates when given to children are depressing to the respiration and for this reason provision should be made to keep the airway patent. Oxygen with a rebreathing bag must be part of the equipment of the operating room when the patient has been given a barbituric acid drug either as a preanesthetic medication or for primary anesthesia.

Local anesthesia assumes an important place in the activities of the peroral endoscopist and he has always been among the first to try out the new drugs that are offered for topical anesthesia. A few of these he has retained but for the most part he continues to use cocaine for topical anesthesia.

Cocaine has been so widely used over such a long period of time that we are more familiar with the action of this drug than we are with the action of some of the more recently described local anesthetics. The accumulated knowledge of the good and evil effects of this substance has done much to make it a safe drug to use. We know that it is a potent drug and we have found no chemical antidote to correct the toxic effects that frequently result from its use. We have learned that individual sensitivity to cocaine is relatively common and that this sensitivity can be anticipated by observing the effect of a small dose during a ten-minute period. Cocaine is not the proper drug for local anesthesia in children up to the age of 15 years. The upper age groups respond well to this drug.

Cocaine for local anesthesia has been used in varying dilutions since it was first used in 1884. Rather than think in terms of dilutions or strength of solution, we should think of the amount of the drug that will be absorbed from the area of the body to which it is applied. Cocaine hydrochloride in 5 per cent strength is a satisfactory and adequate local anesthetic for the routine endoscopic procedure. This is a personal opinion and is offered after having run the gamut of cocaine dilutions from 1 per cent to 20 per cent.

The induction of the local anesthetic should be carried out, when possible, by the operating surgeon. The period of 25 minutes, allotted to the induction, presents an opportunity to establish friendly relations with the patient. It is a matter of great importance in endoscopy that the patient have complete confidence in the surgeon, both as to his veracity and competence. The period of induction will give the surgeon ample time in which to explain to his patient what is

to be anticipated as to pain, discomfort, interference with breathing and after-pain. During the induction period, the surgeon will be in a position to observe any sensitivity to cocaine or early toxic reactions.

The following routine for the induction of local anesthesia for any peroral endoscopic procedure in an adult has been found satisfactory. A 5 per cent solution of cocaine is used. Five cc. of this mixture is placed in a 10 cc. glass syringe to which is added five drops of adrenalin chloride, 1:1000 solution. The syringe serves as a measuring container and is a constant reminder and indicator of how much cocaine has been used and how much remains. At all times it is possible, by viewing the syringe, to estimate how much of the drug the patient has received.

The first step in the induction of cocaine anesthesia of the pharynx, larynx and trachea is to spray the pharynx with the 5 per cent solution, using a hand atomizer and compressing the bulb three times, the solution being distributed over the palate, roots of the upper incisor teeth and the posterior pharyngeal wall. After four minutes, cotton sponges dipped in the solution and the excess expressed from the sponge are applied to the pyriform sinus on either side. These sponges are applied over a period of four minutes. A wait of two minutes now follows to allow for a short reassuring talk to the patient. It is explained that there will be no pain attached to the examination, that relaxation on his part will overcome the sensation of pressure and that his breath will not be shut off. Following these instructions, 2 cc. of the solution are dropped on the vocal chords under mirror guidance. This entire procedure will occupy about 20 minutes and perfect anesthesia should exist and continue for about 30 minutes after the last step of the induction. The solution remaining in the syringe after anesthetizing the larynx is reserved for the bronchoscopic atomizer to control the cough reflex when working on the lower levels of the bronchial tree.

This orderly induction of cocaine anesthesia should be carried out by the surgeon. There are good reasons for this and while it may not be practical in a large clinic, in a small hospital with changing nurse and medical personnel, it is of the utmost importance. The first spraying of the pharynx with an ensuing wait of four minutes allows time, in a patient with unusual sensitivity to cocaine, for toxic symptoms to manifest themselves. These signs and symptoms may be overlooked by nurses and house officers.

Many other local anesthetics have been tried over a period of years but have been abandoned for the more satisfactory cocaine

routine. Novocain has but slight anesthetic action on unbroken mucous membrane. Larocaine in my hands did not produce adequate anesthesia. Sensitivity to pontocaine is frequent and is often followed by constitutional reactions, some of them fatal. A recent report listed three deaths and four toxic reactions following the local use of pontocaine preliminary to bronchoscopic examinations.

The endoscopist who must work in a small hospital with untrained assistants and with changing nurse personnel will find it necessary to resort to general anesthesia for many of his patients. The uncontrollable child, the apprehensive adult, the patient with an impacted foreign body of the esophagus, all cases of this nature will be handled with greater safety if they are in a state of general narcosis.

Of the general anesthetic agents, those of the inhalation group are the best understood. Ether, given by the open drop or closed method, is a safe anesthetic, available to the great majority of patients both young and old. The anesthesia can be continuous throughout the endoscopic examination. For a bronchoscopic examination, the accessory tube on the Jackson type bronchoscope can be made use of to carry ether vapor from a rebreathing bag. When the esophagoscope is used, a flat gauze mask placed over the mouth will permit continued narcosis by the open drop method. A laryngeal examination or operation is of such short duration that it is possible for the anesthetist to bring the patient to a profound depth of narcosis after which the operator will have the necessary two or three minutes to complete his laryngeal examination or operation without additional ether. Ether given in the above manner can be administered by a house officer or trained nurse anesthetist and done with perfect safety.

General anesthesia by means of the barbituric acid derivatives given by the intravenous route finds a wide field of application in peroral endoscopy. The advantages of this type of anesthesia are numerous as compared with other methods of general anesthesia. The induction is rapid and pleasant with an early return of the reflexes, and little or no postanesthetic discomfort. It is possible to obtain complete relaxation and there is no irritation of the respiratory mucosa.

The disadvantages connected with intravenous barbiturate anesthesia are of a serious nature but can be anticipated and partly overcome. The tendency to laryngospasm is always present. The depression of the respiratory and circulatory systems is sometimes pro-

found and terrifying. The barbiturates are potent drugs when given directly into the circulation and the anesthetist must be vigilant and prepared at all times to meet the unpredictable changes that take place during the period of anesthesia.

Pentothal sodium, a barbituric acid derivative, is enjoying current popularity as an intravenous anesthetic. It has replaced evipal, a similar preparation and has many advantages over the latter drug. The induction is pleasant and painless with rapid recovery and there are no disagreeable postanesthetic effects. It has been used for all endoscopic procedures where general narcosis is indicated. Its use is contraindicated in infants, children and the aged.

The prime disadvantage of pentothal as a general anesthetic for any surgical procedure is the frequent appearance of laryngospasm, a serious handicap to any surgical procedure on the air passages. Spasm of the larynx, which is probably due to a hyperactivity of the parasympathetic nervous system, can be controlled in part by careful preanesthetic preparation. The administration of morphine and atropine or scopolamine one hour before the induction with pentothal is essential. Spraying of the pharynx and the larynx with cocaine hydrochloride, 5 per cent solution, allowing 15 minutes for this step in the preanesthetic preparation, is of the utmost importance. With the above preanthesthetic use of morphine and atropine and the topical application of cocaine to the larynx, the incidence of laryngospasm has been infrequent and has never been of such severity that it could not be overcome and controlled by the use of a rebreathing bag with oxygen.

Cullen[2] has been of great assistance to endoscopists in their problems of anesthesia in peroral endoscopy. His recent suggestion as to the use of curare to produce muscular relaxation has been a distinct aid in those cases of muscular spasm. He observes that relaxation of extreme degree can be obtained with relatively little respiratory depression and without noticeable cardiac or vasomotor depression. These observations I have confirmed, especially in cases where there is a spasm of the cricopharyngeus. Entrance to the esophagus has been found to be easy after the use of curare in cases where previous unsuccessful attempts had been made, some under general narcosis. A preparation of curare is now on the market under the trade name Intocostrin.

General anesthesia by the rectal route should be a perfect method of inducing anesthesia for surgical procedures about the air and food passages. Many anesthetics have been used by this route but have

been found to have drawbacks that make them unsatisfactory to both anesthetist and surgeon. Avertin, ether in oil, paraldehyde and the barbiturates have all been used for rectal anesthesia and most of them have been displaced by safer methods.

Avertin, once widely used, first as a general anesthetic and later as a basic anesthetic, now is used infrequently. The respiratory depression, the slow return of the reflexes and the necessity to supplement it with some additional form of anesthesia, all combined to displease the surgeon and bring about a demand for something better. This better method was supplied in pentothal sodium by the intravenous route. All of the barbituric acid derivatives may be given by way of the rectum and this includes sodium amytal, seconal and pentothal. Such use is rare for endoscopic procedures except in the case of infants and children where an opened capsule of seconal or nembutal inserted in the rectum will aid in producing a mild hypnosis and permit an orderly examination of an excitable child.

The endoscopist is forced to use many potent substances for preanesthetic preparation and as anesthetics for his examinations. Most of the drugs used are depressants to the respiratory and circulatory systems. Morphine, cocaine, pentothal and ether can all produce a depression of the respiration to a point where artificial respiration and other corrective measures must be used to restore the respiratory function. With full recognition of the possibility of some of these toxic reactions appearing in any or all cases, the surgeon and the anesthetist will always be prepared to meet these events and since they are prepared, emergencies will seldom arise. Provision is made to counteract toxic reactions from cocaine by the use of one of the barbiturates as preanesthetic medication. In the suggested routine for local anesthesia with cocaine, the pharynx is first sprayed with a 5 per cent solution and a waiting period of ten minutes ensues before the larynx and trachea are anesthesized. During this ten-minute period any peculiar sensitivity will have manifested itself and this will take place before any large amount of the drug has been used. This sensitivity will show itself in the form of pallor, sweating or syncope. Should such signs appear this type of anesthesia is discontinued.

Toxic effects from scopolamine are noted as flushing of the face, incoherence, delirium, or excitability. This is sometimes noted in the older age group and it is in this group that atropine should be used rather than scopolamine. The ill effects of scopolamine may be counteracted by the use of apomorphine gr. 1/40.

Morphine, in the amount used for preanesthetic medication, seldom produces a toxic effect. During the era when it was used as preanesthetic preparation or as a narcotic on children and infants, some alarming toxic effects on the respirations were noted. I have discontinued the use of morphine in children and infants because of a few unfortunate experiences. The attempt at resuscitation of an infant or child with respiratory depression from an overdose of morphine sometimes results in failure and a fatality.

Since pentothal has come to be so frequently the anesthetic of choice for endoscopic procedure where general narcosis is indicated, we have found it necessary to provide for the many unforseen incidents that appear during intravenous anesthesia. Laryngospasm is always expected and for this reason morphine and atropine are always used as the preanesthetic medication. The amount of morphine prescribed is governed by the metabolic activity of the patient. A careful induction of local anesthesia of the larynx and pharynx by means of cocaine, 5 per cent solution, with a waiting period of 15 minutes before the induction of the pentothal anesthesia will help to prevent a spasm of the larynx. This preliminary use of cocaine and the preanesthetic use of morphine and atropine will aid greatly in reducing the number of cases of laryngospasm under pentothal anesthesia. Curare so highly effective as an antispasmodic may be used if spasm is anticipated.

In addition to the problem of laryngospasm there is always the possibility of respiratory depression when potent drugs such as morphine, ether and scopolamine are being used. This depression may result from a disturbance of the central nervous system and dysfunction of the respiratory center. It may follow the partial paralysis of the accessory muscles of respiration. Whatever be the cause, the treatment is to establish a patent airway and provide for inflation and aeration of the lungs. The airway may be obstructed by mechanical means as a foreign body in the pharynx, larynx or trachea. The falling back of the tongue is a perfect example of mechanical interference with the airway as is the state of laryngospasm, this in contradiction to a functional disturbance of respiration as is present with a central respiratory depression or paralysis of the muscles of respiration.

To correct the mechanical interference with the airway, the first step is to withdraw the tongue either by a tongue forceps or gauze covered fingers. A mechanical airway of any of the commonly used types may now be placed in the pharynx or a Magill nasal tube passed through the nose to a level below the base of the tongue. At

times these measures will not overcome an obstruction in the larynx and so it will be necessary to expose the larynx with a speculum and insert a bronchoscope between the vocal cords. After the bronchoscope is in position, oxygen may be insufflated by way of the accessory channel in the Jackson type bronchoscope. It is a simple matter to provide an adaptor of rubber attached to a rebreathing bag and an oxygen supply. If the airway is clear, the bag may be compressed by hand and proper ventilation be obtained. This method of piping oxygen down to the lungs, under proper pressure, by way of a bronchoscope is a most efficient way of providing artificial respiration. It is important that the operator make certain that the airway is clear before compressing the rebreathing bag.

At times during pentothal anesthesia, there are indications of a depressed state of respiration. Respirations may be shallow, infrequent or have ceased. In such a situation, the rubber anesthesia mask can be placed over the face and the rebreathing bag with an oxygen supply attached. By manual compression of the bag forcing air into the lungs, respiration will usually be resumed at once.

Anesthesia is never started until a survey has been completed to assure some responsible person that the following items are available and in good working order in the operating room:

Loaded hypodermic syringes of coramine and adrenalin
Tongue forceps
Magill nasal tubes
Rebreathing bag with face mask and oxygen supply
Mechanical airway
Suction apparatus, with laryngeal and bronchial tips
Laryngeal speculum
Bronchoscope and batteries

Some of these items will be needed in all cases irrespective of the type of anesthesia used. All of them are indispensable when needed and makeshift methods are unsatisfactory when an attempt is made to reestablish breathing when respirations have ceased.

As a recapitulation of our experiences by trial and error with the various methods of anesthesia and preanesthetic preparation of patients who are to submit to endoscopy, the following plan is followed:

Infants do not require anesthesia or preanesthetic preparation for any endoscopic examination or operation. Children from age 3 to 15 years cannot be properly controlled under local anesthesia and

so require general narcosis. This is best obtained by ether, usually given by the open drop method. As preparation for ether anesthesia in a child, a small dose of atropine will do much to check excessive pharyngeal and bronchial secretions. Atropine sulfate, gr. 1/200, for a child of ten years; grain 1/300 for a child of five years makes a good working rule for dosage of this drug in children. Morphine is not given to children.

There will always be a small number of adult patients requiring endoscopic procedures in whom it will be found not advisable to use a local anesthetic. It is in these patients that we find pentothal sodium given by the intravenous route to be only a little short of an ideal anesthetic.

The pre-operative preparation of these patients is of importance. One hour prior to operation morphine with atropine is given. Twenty minutes before operation the pharynx and larynx are prepared with the routine cocaine 5 per cent solution. This preparation should be completed in detail and will result in relaxation of the larynx and cricopharyngeus muscle under the effect of the succeeding pentothal anesthesia. Laryngospasm will not appear if this cocaine anesthesia has been adequate.

SUMMARY

Pentothal has been found to be an anesthetic of high value for peroral endoscopy when a general anesthetic is indicated.

The preanesthetic preparation of the patient with the proper selection of the preliminary medication is of the utmost importance whether the basic anesthesia be of the local or general type.

Preparations, in detail, must be planned to prevent and correct laryngospasm and respiratory depression which may follow the use of pentothal when used as a basic anesthetic.

Cocaine in 5 per cent strength has been found to be the best local anesthetic for endoscopic procedures. A plan for the induction of cocaine anesthesia is described in detail.

750 Main Street.

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XLII

AUDIOMETRY IN THE DIAGNOSIS AND TREATMENT OF DEAFNESS IN CHILDREN

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The entire program of national, state and local child hearing surveys has been directed towards the early detection of hearing losses in children and the subsequent prevention and amelioration of this disability. National and state organizations have appointed committees whose purpose is to discover the number of children with impaired hearing in the public schools and where possible to institute corrective measures for all those with a demonstrated hearing loss. Thousands of children have had their hearing tested in different cities throughout the country. As a result of these surveys, it can be reasonably assumed that three per cent of the children in public schools have a handicapping hearing loss. Rather ironically the children in expensive private schools are not given the advantage of this particular public health study.

In 1926 the multiple or group audiometer was introduced and accepted as a means of screening out school children with impaired hearing. Undoubtedly this technic has served some purpose if only to crystallize interest in hard of hearing children. Its inadequacies are manifold and at the present time scientific otologic thought definitely tends toward abandonment of this method of examination of hearing in children. The reasons for this are fairly obvious. Even with careful check many inaccuracies appear in the results. If an impairment was found, a second test was made and finally to determine the actual hearing loss, a discrete frequency audiogram had to be done. With this type of testing, the youngest group of school children for obvious reasons were automatically eliminated or else the employment of the monitor system was required, a clumsy pro-

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From the Otological Research Laboratory, Abington, Pa.

cedure. The group test does not indicate a beginning high tone loss which is now recognized as an important prognostic factor in children. Though the method made possible the examination of many thousands of children, the results were not conclusive and the prime purpose of such surveys, prevention and alleviation, was not adequately served.

The discrete frequency audiogram gives an immediate and accurate appraisal of the child's hearing level. Obviously fewer children will be examined in any given period of time, but the examination will be critical and conclusive. Special age groups should be selected for examination and in addition all children with an apparent loss and those developing an acute loss should be examined in the course of the year's study. From the medical point of view, it is an essential factor in the final diagnosis. By repeated audiograms alone can the efficacy of any therapy be gauged.

The American Academy of Ophthalmology and Otolaryngology has sponsored through one of its special committees, that assigned for the Prevention and Conservation of Hearing, a program whereby an early diagnosis of a hearing impairment can be made and appropriate measures to correct the hearing loss or prevent further impairment can be instituted. This is the crux of the situation and should be considered the basis of all child hearing studies.

Details of testing technic, referral, and final handling of the cases are of prime importance but of course involve local situations which may not readily conform to a standard plan. This word "standard" is anathema to the majority of otologists. Each one has his own method of compiling and recording data. He considers this his special privilege and prerogative.

In children the otologist is faced with two separate and distinct problems so far as hearing is concerned, the preschool child and the school child of any age. From a chronological standpoint, he must be able to demonstrate the presence or absence of hearing in very young children, those from two to six years of age, and he must be able to determine the extent of the hearing loss in all children of school age. The otologist being a specialist can function only when he is called in consultation. Too often the family doctor or pediatrician attributes retardation of speech development in the preschool child and inadequate achievement in the primary school child to an inherent mental retardation. Parents, school teachers, friends, and relatives are equally at fault. The child is stigmatized. Precious educational years are lost because a hearing impairment has not been

recognized and the child, completely unconscious of his handicap, reacts psychologically in a manner which may warp his entire future life. Time does not permit an adequate elaboration of this problem, but it can be stated without qualification that the problem is a very real and vital one.

The hearing of preschool children can be measured with reasonable accuracy. Any two-year-old child who has not developed speech should be suspect of having impaired hearing. In many young children, two to four years of age, it is essential that the presence of hearing ability be established. This can be done with considerable accuracy by means of the "tone" or "noise field" method, otherwise spoken of as "free field." The child is examined in a quiet or sound proof room and is exposed to the audiometric range of pure tones delivered through a loud speaker, the intensity of the tone stimulus having a range of at least 60 decibels above normal threshold. With even small experience on the part of the examiner, reaction to the stimulus can easily be recognized. There is no fixed pattern of response. In the normal hearing child, response is immediate and unmistakable. In the deafened child, response may be absent at any intensity or may manifest itself by some fleeting change of expression or focusing of attention. These tests have to be repeated. The reaction is an individual one. Few children respond in an identical manner.

The noise field method is even more striking. In the presence of a noise stimulus of broad frequency band, a thermal hiss, delivered at measured intensities, calibrated in 5 decibel steps above threshold, the normal hearing child will react abruptly and promptly when the sound is heard. The reaction of a child with impaired hearing manifests itself at some level between 20 and 60 decibels above threshold. The completely deaf child will fail to react to intensities as high as 100 or more decibels above threshold or a noise intensity approaching the pain level of the normal adult ear.

In this connection mention should be made of the value of the tone or noise field method in the appraisal of hearing in severe cerebral and cerebellar palsy cases. A high percentage of these children have a hearing loss. In the young age group the application of an audiometer receiver to the ear sets off a violent physical reaction which will obscure any specific response to the sound stimulus. In the tone or noise field, the reaction or response is that of an otherwise unhandicapped child. In the less severe cases, a conventional audiogram may be obtained by having an assistant hold the audiometer receiver (Fig. 1).

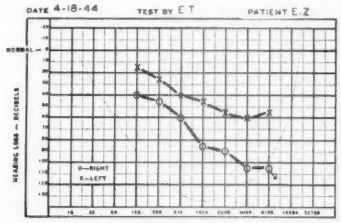


Fig. 1.—Audiogram of patient E. Z., female, aged 13. Diagnosis—cerebral palsy. After examination, she was sent to a school for the deaf for auricular and speech training.

Where circumstances permit repeated tests, young children can be examined with very reasonable accuracy by routine audiometric technics. The receiver is held to the child's ear, response as indicated above is noted at some intensity level for each frequency. The tests may not indicate the actual threshold of hearing, but they do indicate the presence or absence of hearing. Fig. 2 is an audiogram of a child first seen at the age of three and a half years and examined periodically over the four succeeding years. In the last two years she had learned to indicate when the tone was heard. The relative similarity between the first and last audiogram is a matter of interest. The child has never had spontaneous speech and supposedly was born deaf.

Westlake[1] has reported on the examination of the hearing acuity in young children and states that "Children of three and four years of age show less consistency in their response to the pure tone tests and show a wider deviation from accepted normal thresholds than the older ones do, but these are very probably due to other factors than auditory acuity." He further states, "The responses of young children to the audiometric test show considerable reliability." Westlake's studies were made on children with supposedly normal hearing. Quite obviously with increasing age, the accuracy of response will become greater. Under the circumstance, therefore, no question can reasonably be raised as to the value of audiometric examinations in any child of school age.

The establishment of a hearing loss by audiometric examinations is only one phase of the general diagnostic procedure in children with impaired hearing. History and physical examination plus the demonstrated hearing loss are the critical triad. Together they are

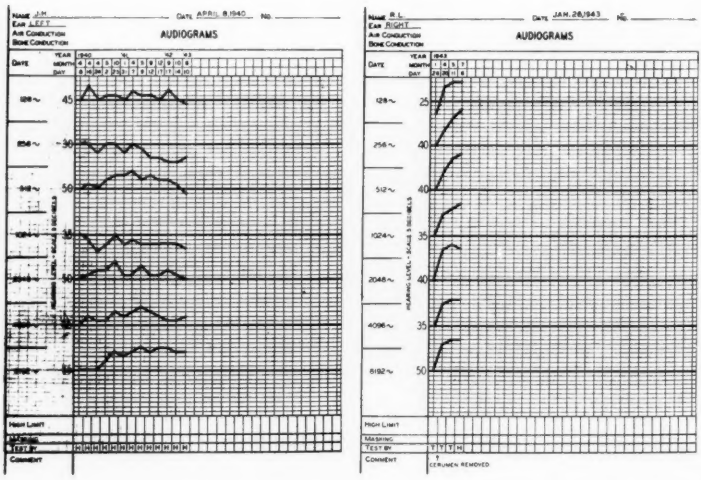


Fig. 2.—Audiogram of patient J. H., female, aged 3½, when first seen. Diagnosis—embryonic deafness. Note general conformity of audiograms over four-year period.

Fig. 3.—Audiogram of patient R. L., male, aged 9. Cerumen at first visit was not removed.

conclusive, individually they are unconvincing. It is assumed that all such children have been given a careful and adequate ear, nose and throat examination and that pertinent facts in the clinical history are a matter of record. Prevention and alleviation of deafness in children is the aim of every otologist and of all groups interested in this important problem. Progress in our knowledge of hearing impairments in children is dependent upon audiometry. No otologic condition in a child should be exposed to treatment until careful appraisal of the actual hearing level has been made.

As described above, reasonably accurate audiograms can be made on children from the age of two years and upward. Special tests for unusual types of hearing losses can be carried out on all children. Masking can be used on children at six years of age or over with great accuracy. Speech reception tests,[2] using properly compiled simple sentences, are probably more accurate measures of hearing in primary school children than audiometer tests. The child's interest is aroused and maintained. There is none of the strain and fatigue of listening

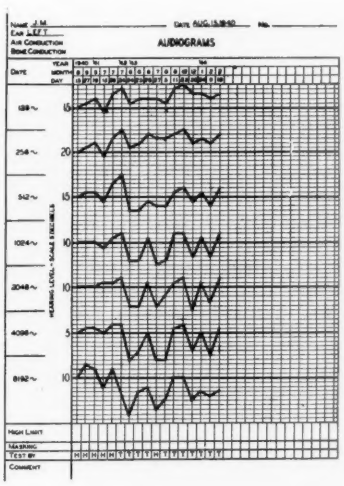


Fig. 4.—Audiogram of patient J. M., male, aged 7, when first seen four years ago. He has repeated attacks of acute otitis, none of which has gone on to suppuration due to sulfathiazole therapy.

Fig. 5.—Audiogram of patient O. M., male, aged 12. Diagnosis—chronic suppurative otitis media. Ear is now dry but perforation has not healed.

for a vague tone signal. The 'game' factor is an incentive in the speech reception test.

The diagnosis of hearing impairment or deafness in children can only be of significance if it points the way towards rational therapy. By the same token the only justification of therapy can be the measured demonstration of improvement in hearing. Subsidence of the acute symptoms of infection of the ear is of course obvious, but the fact that an impairment of hearing is being resolved at the same time can only be accurately demonstrated by audiometry.

At this point the question might be raised whether otologic or audiometric examination should be done first. Ideally, careful otoscopic examination should come first. However, this might not always be feasible, particularly in large groups of children. Under ordinary circumstances, the establishment of a hearing loss would be the indication for referral to the otologist.

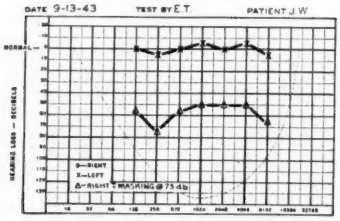


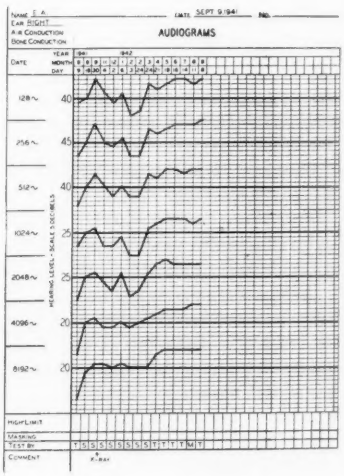
Fig. 6.—Audiogram of patient J. W., male, aged 12, when first seen six years ago. Mastoidectomy, right, done at 17 months of age.

If, fortunately, the canal is found impacted with cerumen, the solution of the difficulty is relatively simple. Such impactions are seldom responsible for a loss of more than 40 decibels. Removal of the cerumen is of course followed by immediate relief of the impairment (Fig. 3).

In the same way foreign bodies will cause an impairment depending on the size, character and duration of domicile of the object. An inflammatory reaction may be present, thus complicating the picture. Lay effort at removal may have traumatized the canal wall or tympanic membrane. Improper handling of these cases may result in serious consequences and this is of fairly frequent occurrence. The insertion of foreign bodies into the canal becomes almost a habit for some little children. Such a history from the parent may be extremely helpful.

By far the most common cause of a hearing loss in children is middle ear infection, acute, subacute or chronic otitis media, suppurative or nonsuppurative. At the present time major concern is directed towards the acute, nonsuppurative type of middle ear infection. It can be controlled by sulfonamide therapy. Subsidence of symptoms and fall in temperature follow sulfonamide therapy in almost routine fashion, but the true measure of successful treatment is a return to normal hearing in the affected ear or ears. All therapy of acute middle ear infection must be controlled by audiometric examination. When a return to normal has been sustained for a period of a week's time, the incident may be considered "closed". Without such evidence therapy and supervision must be continued (Fig. 4).

In the present discussion, mastoid disease and its surgical treatment will be considered only to draw attention to the fact that a re-



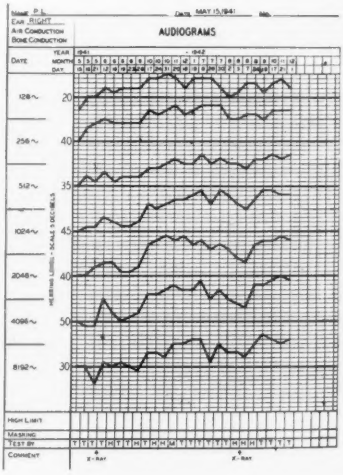


Fig. 7.—Audiogram of patient E. A., male, aged 14, when first seen 3½ years ago. Treatment—x-ray therapy of eustachian tubes.

Fig. 8.—Audiogram of patient P. L., male, aged 14. Treatment—two courses of x-ray therapy of eustachian tubes.

sulting hearing loss is usually permanent and irreversible (Fig. 6). Chronic suppurative otitis media is unhappily still a common condition. It should be completely preventable.

Acute infections of the nasopharynx, sequelae of the common contagious diseases, is preventable with intelligent handling. But the "running ear" is still a matter for the otologist's concern. A great amount of data has been presented relative to the care of these cases by insufflation of iodine powder, of sulfonamide powders or by ionization. The sulfonamides in their microcrystalline form are remarkably effective. However, a therapeutic agent alone cannot be expected to control this situation. The toilet of the ear—proper cleansing—is essential. During such treatment the patient's hearing level must be periodically recorded. Perforations are slow to close if ever, and as they approach closure the hearing level improves or declines, depending upon the condition of the middle ear. Since "hearing" is the requisite, the only purpose of obtaining a dry ear must be measured by repeated hearing tests (Fig. 5).

The persistence of adenoid tissue rests in the posterior nasopharynx, tissue which cannot adequately be removed by surgical

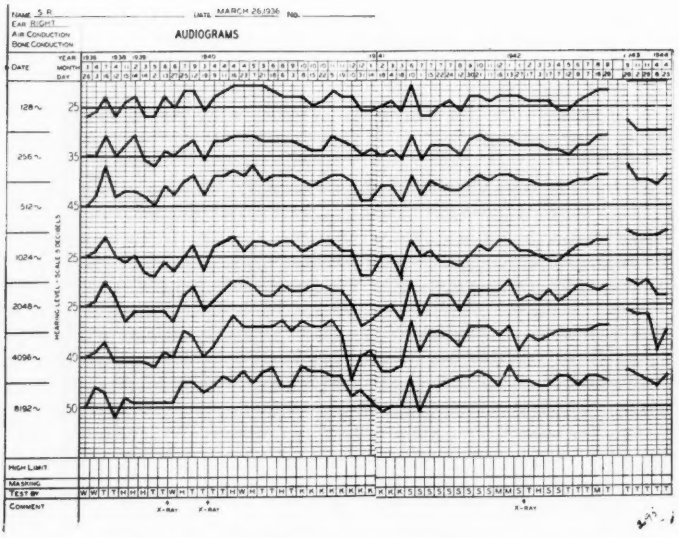


Fig. 9.—Audiogram of patient S. R., male, aged 14, when first seen five years ago. Treatment—three courses of x-ray therapy of eustachian tubes. During a period of five years he had repeated nasopharyngeal infections. Now at 19 years of age, hearing has remained more stabilized.

means, is responsible for a high percentage of aural difficulties in young children. Crowe and his co-workers[3] have focused attention on this condition and have reported at length on the beneficial effect of radon therapy. They have further demonstrated the occurrence of adenoid tissue rests in the eustachian tube itself, which are, of course, quite inaccessible to instrumental removal. The effect of such radiation therapy is striking to a degree. It is one of the outstanding therapeutic measures for the correction of hearing impairments in children.

However, x-ray treatment of this condition can be carried out with equal effectiveness. It would even seem to have some advantages. Though requiring several visits, it is entirely devoid of discomfort and can be used on little children without the necessity of general anesthesia. No untoward results have been observed in a considerable number of children so treated. Figs. 7, 8, and 9 indicate the improvement in hearing obtained in three children who were given six x-ray treatments at one week intervals. Sixty-five roent-

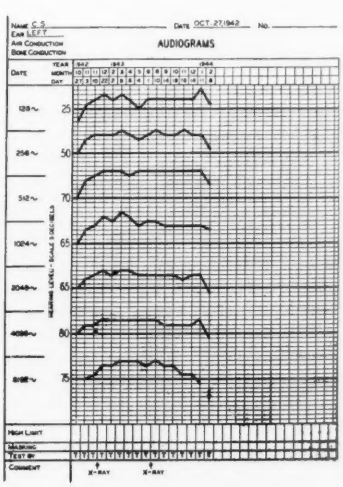
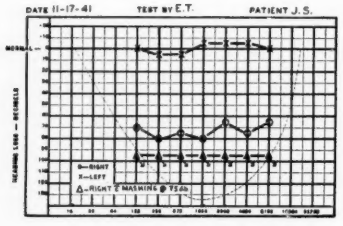


Fig. 10.—Audiogram of patient C. S., male, aged 9. Treatment—x-ray therapy of eustachian tubes. The hearing loss is profound and the tympanic membranes are markedly thickened and retracted. There was improvement in only one ear.

Fig. 11.—Audiogram of patient A. R., female, aged 6. Treatment—tonsillectomy and adenoidectomy. She has maintained improvement without further therapy.

gens are administered over the course of either tube using the following factors: 5 milliamperes; 130 kilovolts; 6 millimeters aluminum filtration; 40 centimeters distance. Over the six weeks' period, 390 roentgens are administered over the course of either tube. With the machine used, 65 roentgens are 10 per cent of a saturation dose. This specific technic has been developed by Dr. J. Donald Zulick, Roentgenologist-in-Chief, of the Abington Memorial Hospital. These cases must be carefully chosen and controlled. All children so treated have previously had tonsillectomy and adenoidectomy performed at least once, adenoid tissue was observed on nasoscopic examination in all cases.

Recovery is not always so striking, as evidenced by the hearing level shown in Fig. 10. The membranae tympani in this case were both markedly thickened and retracted when the patient was first seen and the hearing loss was extreme. There was improvement of hearing in one ear, as shown, none in the other. However, the gen-



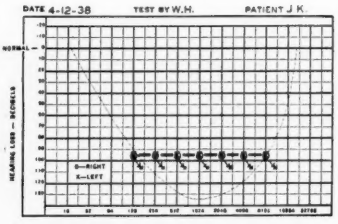


Fig. 12.—Audiogram of patient J. S., male, aged 13. Hearing loss developed during attack of mumps three months prior to audiogram.

Fig. 13.—Audiogram of patient J. X., male, aged 17, showing complete loss of hearing. Diagnosis—reticulo-endotheliosis and xanthomatosis of both temporal bones.

eral hearing level ultimately required placing the child in a school where auricular training and speech correction were available.

Occasionally equally striking results are obtained with tonsillectomy and adenoidectomy alone (Fig. 11). But such improvement can only be safeguarded by careful follow-up audiometric examinations. X-ray or radium therapy is always in reserve if further difficulties develop.

Reference has been made elsewhere[4] to the relationship of unilateral or bilateral hearing loss in children to parotitis. Careful audiometric studies alone can indicate the nature of the loss, a perceptive deafness in every instance (Fig. 12). Following the recognition of a hearing loss in one ear this patient was treated elsewhere with inflation and bouginage for several months. No audiogram was made. When the type of loss was definitely established, the patient could of course be spared further unnecessary and useless treatment. Periodic audiograms throughout the adolescent period serve as a check on the hearing in the unaffected ear in such cases of unilateral deafness.

Fortunately malignancy affecting the ear in children is an unusual condition. Fig. 13 is the audiogram of a boy 17 years of age who had been treated for bilateral furunculosis of the external canals. As indicated there was a total loss of hearing in both ears which had come on gradually. Biopsy revealed reticulo-endotheliosis and xanthomatosis of both temporal bones. Intensive x-ray therapy has controlled the condition completely but the hearing loss remains unchanged. The otic capsules were destroyed.

SUMMARY

In view of the greatly awakened interest of laymen, educators and the medical profession itself in the problems of the hard of hearing child, it has seemed appropriate at this time to emphasize the role of audiometry in the total picture.

The initial phase of the problem was largely a statistical one in which large groups of children were examined in different cities in the country and a relatively uniform morbidity per cent was established. However, follow-up and otologic care of the cases discovered did not keep pace with the primary effort and there ensued a waning of general and specific interest.

Attention has again recently become focused on the problem, largely as a result of the expected number of aural casualties in the armed forces. Here rehabilitation is the primary concern, as indeed it should be in all cases of hearing loss of any degree in civilians, children or adults. Obviously the most fertile field for effective therapy is in the child age group. Hearing surveys now will be carried out with the definite purpose of referring the child through the famliy physician to established otologic clinics where appropriate therapy can be instituted. These clinics will assume a very real responsibility. The care of the children referred must be of the highest order and the data thus obtained should be of great significance in the future development of the therapy of hearing losses in children. Such care is dependent on the exact diagnosis of each individual case and an essential factor in achieving this end is a complete and careful audiometric examination. Too often therapy is considered effective if it arrests further progress of hearing loss. Such cannot be the case in dealing with children. A loss of hearing, except in conditions recognized as irreversible, should be improved. This should be possible in a high percentage of cases and here again the progress of the improvement should be followed. Intelligent therapy can be controlled in no other manner. Following the conclusion of therapy these children should be examined throughout their entire school career to safeguard the gain obtained and to anticipate any recurrence of the difficulty.

CONCLUSIONS

1. Accurate diagnosis of a hearing loss in children is an essential factor in setting up any therapeutic regime, and can be achieved only by complete audiometric studies in conjunction with general and special physical examination.

2. A record of the effect of therapy is essential. Not only are audiograms a measure of success, but they are the only reliable means of indicating the necessity for a change in the therapeutic procedure.

3. Following any type of treatment and regardless of its result, audiometric follow-up should be continued throughout the school life of the child.

ABINGTON MEMORIAL HOSPITAL.

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XLIII

SPHENOID SINUS DRAINAGE

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Chicago, Ill.

Inflammatory conditions involving the sphenoid sinus appear far oftener than is generally supposed. Many cases exist unrecognized, the diagnosis being finally made on the autopsy table.

Teed[1] in an extensive review of the literature previous to 1938, found 129 cases of meningitis traced to the sphenoid sinus. From the statistics collected he concludes that in clinical cases of sinusitis the sphenoid is involved in 15 per cent; in pathologic cases, 33 per cent and yet it is responsible for approximately 35 per cent of all rhinogenic intracranial complications. In these cases the cause of death is usually due to septic meningitis and may be traced to the sphenoid sinus alone or to combined infections which involve the ethmoids or the middle ear.

Courville and Rosenvold[2] studied the records of the 337 cases of intracranial complications found in a series of 15,000 autopsies. In 22 of these the origin of the infection was traced to the sphenoid sinus and in most of these the extension was from an acute inflammatory process.

The prevalence of sphenoid sinus infection may be determined by examination of specimens from cadavers which reveal an occasional thickened irregular mucosal lining indicating infection. In a study of 100 specimens picked at random from the anatomic laboratory, 13 such cases were found.[3] In 10 of these the mucosal lining of the ostium was involved in varying degrees and in some instances the outlet was further blocked by the presence of hyperplastic tissue in the spheno-ethmoid recess.

Szanto[4] obtained strikingly similar results in the study of the sphenoid sinuses examined at autopsy. In 100 consecutive cases one

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Read before the Chicago Laryngological and Otological Society, March 6, 1944, and the Section on Eye, Ear, Nose and Throat of Illinois State Medical Society, at Chicago, May 18, 1944.

or both of the sphenoids were involved in 14 and in 2 of these there was an acute empyema. These findings seem rather surprising to the average rhinologist who seldom searches out and identifies a sphenoid sinus infection. Clinicians are inclined to overlook the posterior cavities and direct their attention to the more accessible sinuses in the anterior group. They investigate the sphenoid only when treatment of the other sinuses fails to bring about relief of the symptoms, or when the symptoms are so pronounced or severe that they demand investigation.

The high rate of incidence, 13 per cent as determined by anatomic study, and 14 per cent as observed at autopsy, would indicate that many of the cases are of low-grade type with minor symptoms. In this group undoubtedly fall those cases of mild postnasal drip, chronic irritative cough and periodic attacks of a husky voice.

In these cases the sinus mucosa is functioning but some interference with drainage prevents a complete cure.

Factors which in the presence of an infection may cause drainage impairment are anatomic and pathologic.

The Anatomic Group:

1. Small ostium, 2 mm. or less in diameter.
2. Ostium near the roof.
3. Encroaching posterior ethmoid cells in the area of the ostium.
4. Shallow spheno-ethmoid recess which permits blockage of heavy secretions.
5. Deviated septum which impairs sinus ventilation and narrows drainage space.
6. Presence of recesses, partial septa and spurs in the sinus walls.

The Pathologic Group:

1. Allergy with or without polyp formation.
2. Thickened inflammatory mucosa which narrows the lumen of the ostium.
3. Hyperplastic tissue, benign or malignant, in the sphenoethmoidal recess encroaching on the ostium.
4. Allied infection in the posterior ethmoids or the opposite sphenoid.

The anatomic factors which may interfere with free drainage from the sphenoid sinus aside from deviated septa are scarcely ever recognized in the living yet are easily analyzed in the study of anatomic material. The ostium may rarely be seen in the living on anterior rhinoscopy and its relations in the spheno-ethmoidal recess are difficult to determine. Some information may be obtained by use of the nasopharyngoscope but more may be gained by probing. Sounding the ostium reveals much concerning its size and location. Further information may be obtained with sinus irrigation. A free return flow indicates an unhampered drainage outlet.

Recesses in the sinus walls may be revealed by roentgen study aided by the use of contrast media and by this means also does one determine the character of the sinus lining membrane and the presence of growths within the cavity.

Symptoms credited to infections in the sphenoid sinus are so multitudinous and variable that they are of little value in assisting in a diagnosis. A satisfactory roentgen study is desirable. This includes the use of contrast media and a variety of views, the most important of which is the submental vertex projection.

Proper investigation also includes mirror examination of the postnasal space. The presence of thick tenacious secretions or crusts in the oro- or naso-pharynx, or on the anterior face of the sinus (as seen by anterior rhinoscopy) are strong indications of the presence of chronic sphenoiditis.

Irrigation of the sinus supersedes all other diagnostic measures in revealing the presence and nature of the infectious process.

Three clinical types of sphenoiditis are observed. They are:

1. Acute empyema.
2. Exacerbation of a chronic infection.
3. Chronic empyema.

The Acute Type. Acute cases are seldom observed or diagnosed as such. Most acute attacks are associated with an allied infection in some other sinus, either acute or chronic, or with an underlying chronic infection in the sphenoid itself.

The following two case reports illustrate: (1) the importance of consideration of the sphenoid in all cases of pansinusitis; (2) the severity of symptoms in an acute attack; and (3) the value of early drainage of blocked cavities in the presence of severe symptoms.

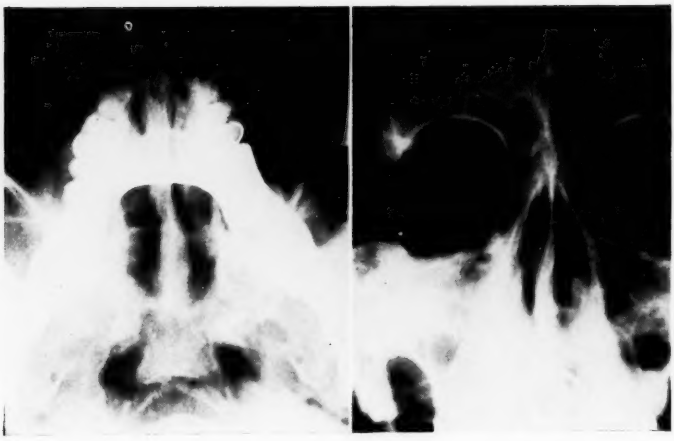

Fig. 1.—Roentgenograms of Case 1. The vertico-mental projection shows moderate opacity of both sphenoid sinuses. In the postero-anterior view the right maxillary sinus shows a marked increased density, pronounced along the walls.

REPORT OF CASES

CASE 1.—J. B., aged 65, for years suffered from recurring attacks of bilateral maxillary sinusitis, both before and after Caldwell-Luc procedures. The maxillary sinuses responded favorably to irrigation through the naso-antral openings until June 1941, when he developed a severe uncontrollable headache not relieved by removal of purulent exudate from the antra. Roentgen study showed a haziness of the ethmoids and sphenoids and displacement therapy was instituted (Fig. 1). By June 18, five days after the onset, the pain was excruciating so a cannula was passed into the sphenoids and these cavities irrigated. Clumps of yellowish exudate were returned from both sinuses and immediate relief followed.

Comment: It was thought at the time that this infection was an extension of the process from the antral infection although there is a strong likelihood that it was merely an acute manifestation of a chronic sphenoiditis which had previously existed unrecognized.

CASE 2.—Miss H. E. R., aged 23, had been treated at various times for over a year for attacks of "sinusitis" and this had included window operations and intranasal frontal sinus drainage. Her symp-

toms persisted and these included a deep-seated headache which became severe periodically. She was first seen at my office May 28, 1942, complaining of severe bilateral frontal headaches. Irrigation of the left frontal sinus and displacement therapy produced mucopurulent clumps and afforded relief. A month later the displacement procedure was repeated a few times with good results.

On July 9, because of persistence of a deep-seated headache, the anterior wall of the left sphenoid was entered and considerable thick, foul-smelling exudate under pressure was removed by irrigation. Pain persisted and that night became so severe that the patient was removed to the hospital where the pain was partially controlled by morphine. Associated with the headache were vomiting, tachycardia and dyspnea. An internist and neurosurgeon saw the patient the next morning and felt that there was great danger of impending intracranial complications or blood stream infection. At that time the temperature was 103° F., the white blood count was 23,150, and the vomiting continued. Sulfadiazine, gm. 4, was given at once intravenously with 2000 cc. of 10 percent dextrose solution. The sulfadiazine was repeated every three hours for four doses and then every four hours for two days. This she took very well and the concentration in the blood never got beyond 5.9. The temperature began going down immediately and in two days it reached 100° F. The pain, however, persisted and on July 14, five days after entrance into the hospital, an opening nearly 1 cm. in diameter, was made in the anterior wall of the sphenoid sinus. This permitted an outpouring of purulent material and was followed by a normal temperature with complete relief from pain. The patient was discharged from the hospital two days later and returned to work the following week.

Culture from the nasal secretions revealed staphylococcus aureus hemolyticus and staphylococcus albus. The blood culture was negative. The patient has been seen at intervals and on one occasion because of a return of the headache, thickened mucosa was removed from the neighborhood of the sphenoid ostium. Since that time several months ago, there has been no return of her symptoms.

Comment: Careful study of the history of this case indicates that the patient undoubtedly had a long-standing infection in her sphenoid which was blocked off by a closed or nearly closed ostium. This infection persisted throughout the period of surgical treatment of the other sinuses and it is possible that proper treatment of the sphenoid early would have forestalled the other surgical measures. The infection as it has existed was undoubtedly of the puriform type with an exudate of lymphocytes and plasma cells. The rapid trans-

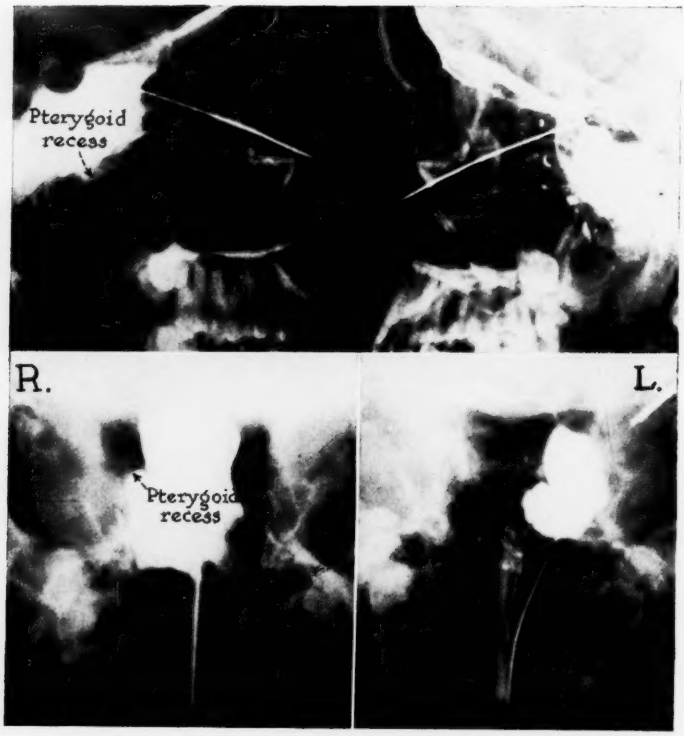

Fig. 2.—Delineation of sphenoid sinuses in Case 3. Lateral and submental-vertex views with iodized oil in the sphenoid sinuses. One week intervened between the study of the two sides. Extensive pneumatization involving the pterygoid process is noted on both sides. No thickening of the sinus mucosa is to be seen in these views.

ition to an active type of infection was induced by trauma of irrigation. The outpouring of polymorphonuclear cells increased the production of exudate far beyond the capacity of the constricted ostium and the symptoms were those of toxic exudate under tension.

The Chronic Type. Symptoms vary from those of a minor nature with a mild postnasal drip and occasional deep-seated headache to those of severe headache and extensive postnasal discharge. Highly diagnostic of chronic sphenoiditis is the presence in the nasopharynx and on the postpharyngeal wall of a heavy tenacious sticky secretion.

REPORT OF CASES

CASE 3.—Miss B. McK., age 28. Years ago she was told that she had catarrh because of the presence of gummy secretion in her throat which was difficult to bring up. For the past three years this was getting worse. She complained of thick material in her throat and at times of becoming feverish and having malaise. A regimen of bilateral sphenoidal irrigations was carried out with positive returns on December 15, 18 and 24, a diminishing return on December 31 and negative irrigation with the pharynx clear on January 18 and 29 (Fig. 2).

CASE 4.—L. P., housewife, aged 55, was first seen April 26, 1943. Five years ago she injured the larynx by a fall and has been hoarse ever since. Her present complaint is attacks of hoarseness with excessive postnasal discharge. Examination revealed a thick, yellowish exudate on the naso-pharyngeal wall and in the interarytenoid space of the larynx. The exudate was also present over the free margin of the cords.

Irrigation of the sphenoid sinuses resulted in the removal of a similar exudate from both sides. Irrigations were repeated at weekly intervals with a gradual lessening of the amount until on the sixth treatment the solution returned clear. At that time her voice was greatly improved and no exudate was discernible in the pharynx and the larynx (Fig. 3).

Comment: The four cases reported here are typical of those usually uncovered in clinical practice. All had been overlooked, yet all had symptoms referrable to the sphenoid sinus.

These cases, though varying in symptoms, type of infection and degree of severity, all responded to the same therapeutic manage-

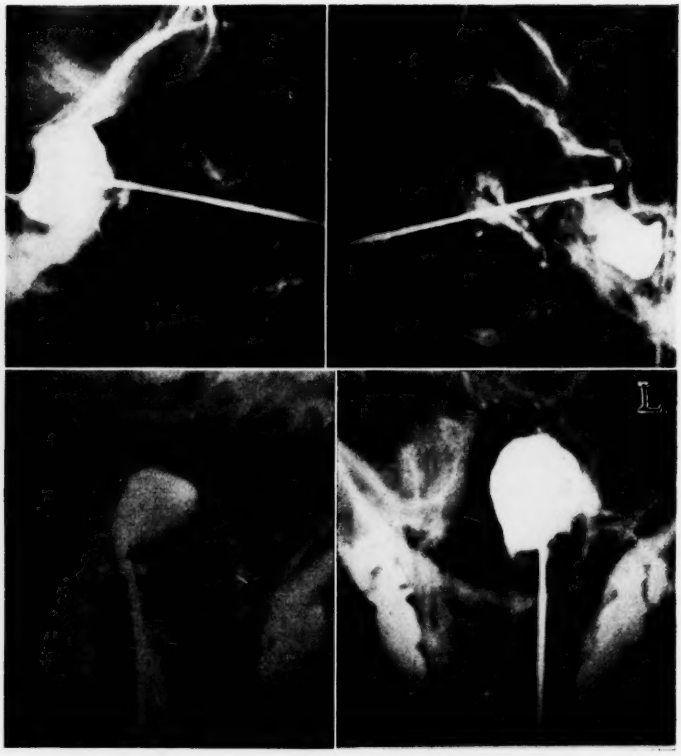

Fig. 3.—The sphenoid sinuses in Case 4. The study was carried out similarly to that shown in Fig. 2. Characteristic overriding of the cavities is noted with thickened mucosa, especially on the right.

ment, namely, the establishment of adequate drainage. In some cases this was accomplished by the mere removal of exudate by irrigation. In one case an enlargement of the drainage outlet was necessary.

All cases do not respond as readily to treatment. Some require further medical and surgical care. In many, an allergy study will be required and the condition may persist until control of the allergy is attained; in others, removal of polyps and hyperplastic tissue from the spheno-ethmoid recess, or the correction of septal defects will be necessary.

The goal, however, is always the same—the establishment of adequate drainage—and this to be accomplished without removal of functioning structures.

Radical procedures which call for the removal of sinus mucosa or the entire face of the sphenoid sinus are rarely indicated and should be instituted only in those cases in which annoying symptoms persist after complete conservative management has failed.

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XLIV

WHAT CAN WE DO FOR MYASTHENIA LARYNGIS?

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It is hoped that this paper will stimulate discussion of the treatment of myasthenia laryngis, a common condition for which we have very little satisfactory therapy.

Myasthenia laryngis is a name applied by Jackson[1] to a morbid entity characterized by asthenia of the phonatory musculature of the larynx, especially the powerful, overworked thyro-arytenoideus muscles. The condition has no connection with myasthenia gravis.

This condition wrecks the careers of a tremendous number of public speakers, actors, and vocalists. It is the most common cause of disability in these professions.

The etiology may be from incessant singing, a predisposition due to congenital defects overloading the phonatory muscles, direct or indirect trauma from conversation, myositis incidental to acute, infective laryngitis, hyperplasia in chronic laryngitis, excessive fatigue, vocal nodules, hematomas, other tumors, arthritic impairment of mobility, and defects in innervation.

Diagnosis of the condition is of course a prerequisite for proper therapy. Chronic laryngitis is frequently mistaken for myasthenia laryngis. Chronic laryngitis may or may not coexist. We must eliminate malingering, diseases of the recurrent laryngeal nerves, tumors in the larynx proper, tumors impairing the function of other muscles of phonation, myositis, arthritis, diseases of the laryngeal cartilages, and all organic lesions.

Prophylaxis is the essence of successful treatment of myasthenia laryngis, as a cure can be obtained only in the early stages of the disease.[1] Any suspect should be instructed to eliminate all possible strain on the phonatory musculature. The laryngologist should explain that there has to be a period of complete rest, followed by no excessive singing or talking. By "complete rest" is meant absolute

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physical quiet and silence. Instructions should be given for voice control. The range of the singing voice extends only about two octaves, seldom three; the pitch of the speaking voice varies for the purpose the individual speaker wishes. He may desire great emphasis and thus strain to a very high pitch, or shriek. If an orator can be convinced that a logical appeal to the intellect of his audience has more value than great vehemence in the voice, then an intractable myasthenia laryngis can be avoided and his career prolonged. Likewise, if the singer can be convinced that it is better to have a good, and permanently good, voice rather than to lose his best tonal qualities by straining to extend the pitch beyond his safe limits, his voice can be preserved.

Sometimes a patient subject to no vocational strain complains of recurrent hoarseness, tired voice, loss of tonal quality and partial or complete loss of voice. If we can eliminate the contraindications outlined above and a mirror view of the larynx reveals that the chink of the vocal cords is elliptic with flaccid margins or that a spastic condition is present, we again have myasthenia laryngis. Such patients usually talk, smoke, or drink excessively. They often have confusing physical handicaps indicating a need for general therapy.

The author first became particularly interested in myasthenia laryngis in 1931 when a very close doctor friend sent his son for treatment to preserve the boy's voice for the final state declamation contest which was to be held in a few days. Because this is a typical case and the exact type the author has termed myasthenia laryngis, his symptoms and treatment are given in detail.

The boy, a senior in high school, had been the winner in many declamation contests during his four years in high school. He had been taught to raise the pitch of his voice to the extreme for emphasis, a method employed by many teachers. He also had been trained to rehearse excessively. About the time he won the semifinals his voice gave away. Both the boy and his family thought complete rest for his voice was out of the question. He was above the average physically as well as mentally and aside from myasthenia laryngis showed no other signs of physical impairment or fatigue. With great effort the boy completed his last contest but lost his voice for three weeks thereafter. During this time he was unable to talk above a whisper.

This experience, however, was not enough to convince him that he must stop using his voice excessively and in such a high pitch. He continued his declamation and public speaking in college. After four years of this, his ordinary conversational voice had a rasp, was subject

to an adolescent cracking, and it was an effort for him to declaim. He came to see me again before he started to medical school with almost complete loss of phonation. He was advised to be absolutely silent and have general rest until he could begin to talk in a whisper without any discomfort. The details of how to use his voice following this rest period were carefully covered again.

This patient reported at weekly intervals until his vocal mechanism gave a partly normal mirror appearance; that is, the glottic chink lost its elliptical appearance. He went through medical school with no further abuse of his voice. However, during his internship, he was called upon to make public talks, and he used his voice excessively in taking patients' histories. The old trouble recurred. Another period of complete rest plus more sunshine and recreation finally restored phonation.

A recurrence at the beginning of a three-year fellowship in surgery was the occasion for the same advice as to diagnosis and treatment. This time the instructions were followed rigidly.

Recently when this doctor was at home on a furlough, he told me that as long as he did not talk excessively and did not try to raise the pitch of his voice, he had no trouble. If he had controlled the use of his voice by the proper regimen when he first began to have trouble, he could have continued with his useful public speaking.

On young individuals, the author has for several years discontinued the use of supportive and especially medicinal help, such as nitric acid and lump sugar, that are used during and a few minutes before the use of the voice. This has been done because it is believed that no help should be given the younger individual inasmuch as this apparent aid is in reality a great harm, due to the fact that the patient is less apt to be convinced that his only cure is prophylaxis in the early stages of the trouble. However, it is thought that an older individual has the right to choose between the permanent loss of his voice or the drastic regimen needed to preserve it.

Another young individual with myasthenia laryngis came to me for treatment of her voice in January 1935. This young lady taught music in our public high school, trained and led a church choir and, in addition, she sang frequently at various entertainments. Even when she was not singing, she used her voice excessively in incessant conversation. There was no physical impairment of any type except the difficulty of controlling her voice as she had previously been accustomed to doing. She was beginning to lose confidence in her ability to sing. She could not reach a "high tone" or "hold a

tone" without twice her usual effort. When she was first seen, she was not in real trouble; but she continued to use her voice all day in demonstrating to pupils, in singing frequently in public, and in talking incessantly, and in two years she came back with a marked impairment in her ability to sing. She was instructed to completely rest her voice for three months. Although she thought this impossible, she did consent to a two weeks' rest period and thereafter for three months to discontinue all public singing and conversation except in school. Her improvement was immediate, but her original ability in singing could not be restored.

Another patient, a lawyer, was seen the first time in 1930 for loss of his voice following a long litigation. He had always been an incessant, rapid talker. His physical condition was perfect in every respect except for myasthenia laryngis. He was a brilliant individual, as well as an expert tennis and football player, with letters in both sports in high school and college. The trouble with his voice became evident soon after he began the practice of law. Each prolonged, excessive argument in a difficult trial would cause a recurrence of aphonia or near aphonia. He quit the practice of law in 1935. From then on he did not have any severe recurrence, although due to his incessant talking he never regained his normal voice, and at times it was an effort for him to speak.

We have now considered myasthenia laryngis in the public speaker, the singer, and lawyer. It is of common occurrence in these three professions particularly where the individual is also an incessant conversationalist.

Can we consider the condition due to an innervation disturbance or muscular fatigue, or both? The author has not been able to eliminate innervation pathology nor prove its presence. There are so many accessory mechanisms to phonation that nerve involvement would surely be manifest elsewhere if present. The author eliminates myasthenia gravis as having any connection with myasthenia laryngis because it ceases to be the latter when an associated organic lesion affecting other parts exists, in his opinion, unless myasthenia laryngis can be demonstrated to coexist and occur without the presence of the organic lesion.

In treating myasthenia laryngis, the author cannot agree with certain contemporary doctors in their use of prostigmin as is used in the treatment of mysthenia gravis.[2] However, the proper treatment for myasthenia gravis is of course instigated whenever it is recognized. It is the practice of the writer to treat myasthenia laryngis in

the manner already covered and, in addition, to give instructions for exercises for relaxation of the phonatory muscles. Yawning or the motion of yawning relaxes the muscles of the throat and mouth. After deep inspiration, one is told to exhale gradually through a small smoking pipe stem. With the chest held forward as much as possible, very deep deliberate breathing should be done for ten breaths at a single series. A very good exercise for resting the tired muscles of phonation is to change the pitch of the voice to another pitch just higher, but not much, than the one in use. We are all prone to talk in too low a pitch. If one is reading aloud, the voice may become fatigued; a slight change of pitch will give immediate relief. Voice students should be impressed with the importance of light staccato vocalizing in developing the musculature for singing.

A patient can be impressed with the degree of effort required to talk or sing by asking him to exhale gently with his lips in a position to whistle. Then he is instructed to increase the intensity of the expelled air enough to make a sound or a musical tone.[3, 4] One can be impressed with the amount of trauma to the larynx incident to talking by placing the thumb and forefinger on the thyroid cartilages first when he whispers and again when he talks. Practically no vibration is felt during a whisper, but definite vibrations are felt during speech.[5]

Students should be taught the proper use of the middle register in singing and speaking. Either a very high or a very low pitch should be avoided. The comparatively few indivduals who approach and sustain very high or very low tones with ease because of their particular vocal mechanisms are excepted of course from this general statement. Composers of music for children and instructors for public speaking should be impressed with the necessity of conforming to this rule in order to prevent myasthenia laryngis. A writer's cramp can be relieved by rest, but myasthenia laryngis is more like the athlete's heart; once it is present, it is there to stay. A status quo is the best we can hope for in most cases. Prevention is the only cure.

CONCLUSIONS

(1) Myasthenia laryngis is a name applied to a morbid entity characterized by asthenia of the phonatory musculature of the larynx, especially the powerful, overworked thyro-arytenoideus muscles. It has no connection with myasthenia gravis.

(2) Constant, excessive use of a very high or a very low pitch is the primary cause for myasthenia laryngis.

(3) Given a complaint of recurrent hoarseness, tired voice, and loss of tone, with a mirror view of the larynx showing an elliptic chink in the vocal cords, we can make a diagnosis of myasthenia laryngis provided we have eliminated malingering, diseases of the recurrent laryngeal nerves, tumors in the larynx proper, tumors impairing the function of other muscles of phonation, myositis, arthritis, diseases of the laryngeal cartilages, and all other organic lesions.

(4) Prophylaxis is the essence of the successful treatment of myasthenia laryngis as a cure can be obtained only in the early stages of the disease. The author has had no beneficial effects from the use of prostigmin.

(5) Relaxation of the muscles of phonation by special exercises is of great value for preventing a recurrence, but prolonged absolute silence and rest must always precede any treatment.

(6) The middle register must be used to the exclusion of constant very high or very low pitches in singing or in talking.

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XLV

TUBERCULOSIS OF THE NASOPHARYNX

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AND

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Tuberculosis of the nasopharynx is only meagerly described in textbooks on pathology and otolaryngology. In fact, perusal of a number of standard works revealed but casual reference to this condition. Its frequent incidence and relationship to pulmonary tuberculosis render it a problem meriting more serious consideration than has been accorded it in the past. Our interest was constantly increased as we proceeded with a general pathologic study of 134 autopsy specimens of the pharynx. To our knowledge there is no record in English medical literature of any investigation on as large a collection of autopsy material of this organ.

Fraenkel,[1] in 1881, studied 50 specimens of the nasopharynx from patients who died of tuberculosis. In ten he found tuberculous ulcers. In eight of these the ulcers involved the pharyngeal tonsil, and in two, the torus tubarius. In 1893, Dmochowsky[2] examined the nasopharyngeal cavity of 64 autopsy subjects with pulmonary tuberculosis and found grossly tuberculous ulcers in 21 specimens. In a study of tuberculosis in infants Ghon and Roman,[3] in 1913, reported 13 instances of nasopharyngeal tuberculosis.

Gräff,[4] in 1934, examined the nasopharynx in 43 subjects with lung tuberculosis and determined by careful histopathologic studies tuberculous changes in 32 specimens (82 per cent). This convinced him of the high incidence of nasopharyngeal tuberculosis in patients with cavernous pulmonary tuberculosis. Gräff stressed the importance of attention to the nasopharynx as a factor in the course and prognosis of the pulmonary involvement. He speculated that even after successful pneumothorax therapy, tubercle bacilli may be

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aspirated from coexisting tuberculous ulcers of the nasopharynx and thus produce reinfection of the lungs.

While routine autopsy examinations of the nasopharynx have been performed by few investigators, histopathologic study of the adenoids removed surgically has been done by several workers. White,[5] in 1907, examined histopathologically 75 specimens of adenoids and in five per cent of them found tuberculous granulation tissue characterized by epithelioid cells, giant cells and caseation necrosis. He believed that the adenoids were the primary site of the tuberculous infection with secondary involvement of the cervical glands. Lachmann,[6] in 1908, likewise observed tuberculous changes in the adenoids in 4.3 per cent of the specimens he examined. The view expressed by White is subject to serious criticism, since the formation of "tubercles" in the adenoids is frequently initiated by the reaction of this tissue to foreign bodies.

Dietrich,[7] in 1916, briefly discussing tuberculosis of the nasopharynx, differentiated tuberculosis of the mucous membrane from tuberculosis of the nasopharyngeal tonsil. He considered primary tuberculosis of the mucous membrane of the nasopharynx as a very rare occurrence. He described a case of a large tuberculous ulcer of the nasopharynx which involved the atlas secondarily. This patient also had pulmonary tuberculosis, but in Dietrich's opinion, the pathologic changes in the nasopharynx were older than those in the lungs. Involvement of the nasopharynx by the hematogenous route was not given much credence.

Trenis,[8] in 1940, reported two cases of tuberculosis of the nasopharynx with positive sputum findings and negative roentgen examination of the chest. One of the patients was an 18-year-old girl with bilateral enlarged cervical glands. The smear was positive for tubercle bacilli. Laryngoscopic examination was negative. When, however, the nasopharynx was investigated, it revealed a white exudate in the adenoid area. The adenoid tissue was removed. Histopathologically it presented numerous epithelioid tubercles with caseation necrosis. After operation, the tubercle bacilli disappeared from the sputum and the patient made an uneventful recovery.

The second patient was a 28-year-old male with Hodgkin's disease of the lung and mediastinum. While there was no tuberculosis of the lungs or larynx, the sputum was positive for tubercle bacilli. Examination of the nasopharynx revealed an ulceration in the adenoid area. Biopsy disclosed numerous distinct confluent epithelioid tubercles with caseation necrosis. Both of these patients asserted that the sputum was not coughed up from the lungs.

Fig. 1.—Histopathologic section of posterior part of roof of nasopharynx from 65-year-old male with fibrotic type of pulmonary tuberculosis.

There were 24 subjects with various forms of tuberculosis in the series comprising the material for the present investigation (Table 1). The complications as observed by autopsy examinations are set forth in Table 2. Of special interest is the fact that the nasopharynx was involved in 18 specimens (75 per cent). Gross lesions were visible in 14 instances, while in 4 the lesions were detected only by microscopic examination. Significant in one subject with proliferative fibrosis of the lungs were the gross tuberculous changes in the nasopharynx. Of the subjects with fibrotic pulmonary tuberculosis, one finally revealed histologic changes in the nasopharynx characteristic of tuberculous disease (Fig. 1).

It may be gathered from Table 1 that common sites of tuberculous lesions of the nasopharynx are the posterior part of the roof and the upper part of the posterior wall. Less frequently tubercu-

Fig. 2.—Gross specimen of nasopharynx; *A.* Septum nasi; *B.* Remnants of pharyngeal tonsil; *C.* Ostium of eustachian tube; *D.* Rosenmueller's recess; *E.* Tuberculous ulcer; *F.* Tuberculous ulcer of larynx.

lous lesions are detected at the posterior margin of the ostia of the eustachian tubes and the nasopharyngeal surface of the uvula and the soft palate.

The gross pathologic changes were characterized by irregular ulcers, mostly ovoid in shape, with undermined margins and yellowish-gray bases. Of the specimens which were grossly negative, histopathologic examinations disclosed in four instances subepithelial tubercles consisting of epithelioid cells, giant cells, and incipient or advanced caseation necrosis.

As previously stated gross tuberculous lesions were found in 14 of our 18 specimens (Fig. 2). In four the lesions were detected by microscopic examination. In Gräff's studies of 118 subjects with open, active tuberculosis of the lungs, lesions in the nasopharynx were observed grossly in 36 per cent of the specimens. When histopath-

ologic study was resorted to in 43 specimens in which gross findings were negative, 82 per cent showed tuberculous changes in the nasopharynx. Both Gräff's results and ours point to the inadequacy of gross inspection for a conclusive diagnosis.

There are two types of tuberculosis of the nasopharynx: (1) the open ulcerous, and (2) the closed (grossly invisible). Obviously, unless microscopic study is resorted to in all instances, one frequently will fail in diagnosis of the closed type.

Tuberculosis of the nasopharynx as a complication of pulmonary tuberculosis has received too brief mention in the medical literature. While our data do not permit us to state that the nasopharynx may be a primary site of tuberculous infection, they are convincing of the frequency with which this organ may become involved in the course of pulmonary tuberculosis.

The pathogenesis of tuberculosis of the nasopharynx may be explained on the same theory as tuberculous disease of the intestines—inoculation of the mucous membrane by contact with sputum containing tubercle bacilli. This perhaps accounts also for the localization of ulcers, in most cases, on the posterior part of the roof and on the posterior wall of the nasopharynx.

While the deep inferior cervical lymph nodes showed the usual enlargement, such a change was not observed in relation to the superior cervical and the retropharyngeal lymph nodes.

In our two cases of miliary tuberculosis, the gross and the microscopic findings were negative for tuberculosis of the pharynx. This would seem to favor Dietrich's view that infection by the hematogenous route is rare.

Clinically it is essential to know the common sites of localization of tuberculous lesions of the nasopharynx. These sites have already been described.

In a number of our specimens the nasopharyngeal and the tubal tonsils showed a partial or complete involution. The presence or absence of tuberculosis of the nasopharynx is independent of the developmental stage of the nasopharyngeal lymphatic tissue.

None of our specimens displayed any microscopic evidence of tuberculous involvement of the middle ear, despite the presence of tuberculous ulcers around the ostia of the eustachian tubes.

Tuberculous disease of the nasopharynx in the course of fibrotic tuberculosis of the lungs is of more practical significance than the fre-

quent incidence of tuberculosis of the nasopharynx in far-advanced fibrocaseous ulcerative tuberculosis of the lungs. It is not farfetched to assume that the nasopharynx may be the persistent source of reinfection of the lungs. Furthermore, it is not an unlikely source of a positive sputum in arrested pulmonary tuberculous disease.

It is significant that our findings and those of Gräff are practically the same. The only difference is that the tuberculous process was found by him grossly in 36 per cent of his specimens, while in our series gross changes were observed in 58.3 per cent. This variation may be explained, however, by the fact that his material was obtained from the tuberculosis wards of a general hospital, while ours consisted of subjects with far-advanced pulmonary tuberculosis from the wards of mental institutions.

Our studies emphatically demonstrate the value of a clinical investigation of the nasopharynx in all routine physical examinations, especially in persons with pulmonary tuberculosis. Certainly, an examination of this organ is quite as important as that of the larynx. In the absence of other findings an obscure focus may often prove to be present in the nasopharyngeal cavity.

SUMMARY AND CONCLUSIONS

1. Tuberculosis of the nasopharynx has been a neglected medical problem. As a complication of pulmonary tuberculosis its incidence is greater than that of tuberculosis of the larynx (Table 2).

2. In 134 consecutive autopsies on which a general pathologic study of the nasopharynx is being performed, there were 24 subjects with various forms of pulmonary tuberculosis, and of these, 18 (75 per cent) showed tuberculous involvement of the nasopharynx.

3. Histopathologic study is often essential in rendering a positive diagnosis of tuberculosis of the nasopharynx.

4. The nasopharynx may be the site of a persistent focus of tuberculous infection, and this may frequently explain the source of reinfection of the lungs in persons who have previously experienced an arrest of pulmonary involvement.

5. A plea is made for careful, routine clinical investigation of the nasopharynx in all rhinolaryngologic examinations, especially in patients with pulmonary tuberculous disease.

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TABLE 1

Data Concerning Tuberculosis of the Nasopharynx in 24 Subjects with Pulmonary Tuberculosis

Case No.	Age	Sex	Race	Anatomic Diagnosis	Type of Tuberculous Lung Involvement	Extrapulmonary Tuberculosis	Nasopharynx
2	65	M	W	Broncho pneumonia; bronchiectasis; meningioma	Fibrotic, apical regions	Ulcer of larynx	Remnants pharyngeal tonsil; grossly negative for tuberculosis; microscopically, subepithelial tubercles with caseation
6	31	F	W	Fibrocaseous ulcerous pulmonary tuberculosis	Fibrocaseous ulcerous	Ulcer of larynx and palatine tonsils	Ulcer on roof; complete involution pharyngeal and tubal tonsils
12	59	F	W	Fibrocaseous tuberculosis of lungs, intestines and larynx	Fibrocaseous	Intestines, larynx and palatine tonsils	Partial involution pharyngeal and complete involution tubal tonsils; multiple ulcers roof and posterior wall; ulcer posterior lip eustachian ostium
13	67	F	W	Fibrocaseous pulmonary tuberculosis; gangrene of left foot	Fibrocaseous		Grossly, almost complete involution pharyngeal tonsil; complete involution tubal tonsil; subepithelial tubercles with giant cells and caseation only on microscopic examination

TABLE 1—(*Continued*)

Case No.	Age	Sex	Race	Anatomic Diagnosis	Type of Tuberculous Lung Involvement	Extrapulmonary Tuberculosis	Nasopharynx
14	56	M	N	Fibrocaseous tuberculosis of lungs, intestines, peritoneum	Fibrocaseous	Peritonitis	Tuberculous ulcer nasopharyngeal surface uvula; complete involution nasopharyngeal and tubal tonsils
22	36	M	W	Fibrocaseous tuberculosis of lung, intestines	Fibrocaseous	Intestines	Pharyngeal tonsil preserved; tubal tonsil almost complete involution; ulcer anterior margin right eustachian ostium
27	50	F	W	Fibrocaseous tuberculosis of lungs; tuberculous peritonitis, salpingitis, cervicitis; amyloidosis liver, kidneys, spleen	Fibrous proliferative	Salpingitis, peritonitis, endometriosis, cervicitis	Grossly and microscopically negative for tuberculosis
28	58	M	W	Fibrocaseous ulcerous pulmonary tuberculosis	Fibrocaseous		Complete involution, nasopharyngeal and tubal tonsil; ulcer nasal surface uvula, roof; tubal elevation, left side

TABLE 1—(*Continued*)

Case No.	Age	Sex	Race	Anatomic Diagnosis	Type of Tuberculous Lung Involvement	Extrapulmonary Tuberculosis	Nasopharynx
29	44	M	N	Fibrocaseous pulmonary tuberculosis; tuberculosis peritonitis	Fibrocaseous	Peritonitis	Remnants pharyngeal tonsil; ulcer nasal surface uvula, soft palate; right tubal elevation
30	57	M	W	Fibrocaseous pulmonary tuberculosis; gangrene of both feet; tuberculosis of larynx	Fibrocaseous	Larynx	Involution pharyngeal and tubal tonsil; small pharyngeal bursa; grossly and microscopically negative for tuberculosis
37	32	M	W	Fibrocaseous pulmonary and intestinal tuberculosis; empyema left pleural cavity	Fibrocaseous		Remnants pharyngeal tonsil; complete involution tubal tonsil; grossly negative for tuberculosis; microscopically, subepithelial tubercles with giant cells and caseation
46	72	M	W	Hypertrophy left ventricle heart; arteriosclerotic contracted kidney; passive congestion liver	Old fibrotic, left upper lobe with caseation subapical region		Ulcer 1 x 2 mm. diameter on left tubal elevation; ulcer upper part posterior wall, left from midline

TABLE 1—(*Continued*)

Case No.	Age	Sex	Race	Anatomic Diagnosis	Type of Tuberculous Lung Involvement	Extrapulmonary Tuberculosis	Nasopharynx
48	64	M	W	Miliary tuberculosis			Negative for tuberculosis
50	48	M	W	Fibrocaseous tuberculosis of lung, peritoneum, mesentery lymph nodes	Fibrocaseous	Tuberculosis peritoneum and mesentery lymph nodes	Pharyngeal tonsil present; on roof, two small tuberculous ulcers; one behind eustachian ostium
54	62	F	N	Fibrocaseous pulmonary tuberculosis	Fibrocaseous		Tuberculous ulcer 2 x 1 mm. on roof, left from midline; small one on nasal surface soft palate; complete involution pharyngeal tonsil
57	29	M	W	Fibrocaseous ulcerous tuberculosis of lungs, larynx, intestines	Fibrocaseous	Larynx, intestines, right palatine tonsil; ulcer in right pyriform recess	Involution pharyngeal and tubal tonsil; two ulcers posterior part roof; one, posterior margin eustachian tube and nasal surface uvula
63	95	F	N	Fibrocaseous ulcer right lung; tuberculous osteomyelitis of sternum		Osteomyelitis of sternum	Negative for tuberculosis

TABLE 1—(*Continued*)

Case No.	Age	Sex	Race	Anatomic Diagnosis	Type of Tuberculous Lung Involvement	Extrapulmonary Tuberculosis	Nasopharynx
64	81	F	N	Coronary sclerosis; myocarditis; fibrotic pulmonary tuberculosis	Bilateral fibrotic		Negative for tuberculosis
82	48	M	W	Fibrinous serous pleuritis; proliferative tuberculosis right upper lobe	Fibrinous serous pleuritis, proliferative (guinea pig inoculation positive with pleural exudate)		Hyperplasia pharyngeal tonsil; negative for tuberculosis
89	28	M	W	Miliary tuberculosis	Miliary		Complete involution tubal and pharyngeal tonsil; negative for tuberculosis
90	31	M	W	Fibrocaseous tuberculosis of lungs, intestines	Fibrocaseous	Intestines	Partial involution pharyngeal tonsil; tuberculous ulcer nasal surface uvula and soft palate; tuberculous ulcer roof anterior lip eustachian ostium
100	52	F	N	Fibrocaseous tuberculosis of lungs, intestines	Fibrocaseous	Intestines	Partial involution pharyngeal tonsil; tuberculous ulcer nasal surface uvula, soft palate, roof anterior lip eustachian ostium

TABLE 1—(*Continued*)

Case No.	Age	Sex	Race	Anatomic Diagnosis	Type of Tuberculous Lung Involvement	Extrapulmonary Tuberculosis	Nasopharynx
109	46	M	N	Fibrocaseous tuberculosis of lungs; osteomyelitis 5th rib; tuberculosis of left shoulder joint; empyema sphenoid sinus; generalized amyloidosis	Fibrocaseous	Osteomyelitis; ulcer on left palatine tonsil	Remnants pharyngeal tonsil; tuberculous ulcer posterior wall
110	42	F	W	Fibrocaseous ulcerous tuberculosis lungs, intestines, larynx	Fibrocaseous	Intestines, larynx	Remnants nasopharyngeal tonsil; tuberculous ulcer posterior wall

TABLE 2

Incidence of Extrapulmonary Tuberculous Complications in 24 Subjects with Pulmonary Tuberculosis

	Number	Tuberculous Involvement of the Nasopharynx: Gross Tuberculous Changes	Tuberculous Involvement of the Nasopharynx: Histologic Changes Only	Larynx	Intestines and/or Peritoneum	Palatine Tonsil (Gross)
Far advanced fibrocaseous ulcerous tuberculosis of lung	18	13	3	5	11	3
Fibrotic type of tuberculosis of lung	2	—	1	1	—	—
Fibrotic type with beginning fibrocaseous changes	1	1	—	—	—	—
Fibrotic tuberculosis with exudative pleuritis	1	—	—	—	—	—
Miliary tuberculosis	2	—	—	—	—	—

XLVI

OSTEOMYELITIS OF THE FRONTAL BONE TREATED WITH PENICILLIN

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With the advent of penicillin to our therapeutic armamentarium, it would appear that the field of otolaryngology may also derive some share of the benefits of this new drug.

It is, however, with great hesitation and not without some apprehension that I present two cases of acute, frontal sinusitis with complicating osteomyelitis of the frontal bone, both of which were treated with this drug, and a third case which was somewhat differently treated for comparison. I say this advisedly because these cases are still under observation and care. However, the encouraging results obtained thus far would indicate that this method of treatment bodes well to supersede our previous methods, in which severe deformity has resulted.

These cases were treated with conservative surgical measures and the greatest dependence was placed on the penicillin, with the cooperation and guidance of our Penicillin Officer, Captain Albert L. Evans.

It would perhaps be germane to present a brief résumé of penicillin therapy as we know it today. Between April and June, 1943, the Office of the Surgeon General of the United States Army sponsored units for penicillin therapy at two general hospitals. These units functioned as schools in penicillin therapy for selected medical officers, who in turn were designated to other installations to carry on the work. At each post where these studies were made, a ward unit was thereby set aside for penicillin therapy under the direction of such a trained medical officer and the chief of the surgical service.

Penicillin has been employed, both as a calcium and a sodium salt, but our experience has been mainly with the sodium salt, which occurs as a fine, orange powder, which is very soluble in water, in

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physiological sodium chloride solution or in five per cent dextrose solution.

The effectiveness of penicillin has been mainly in the treatment of infections caused by the gram-positive organisms; it is not effective against gram-negative organisms, except the gonococcus and meningococcus.

Penicillin may be administered intravenously, intramuscularly and by local application, and usually all three methods are employed in each case. The concentration for intravenous use is 1,000 Oxford units per cc. and the rate of injection varies from 5,000 to 10,000 units an hour day and night, when this method alone is used. However, when all three methods are employed, there may be merely one initial intravenous injection of 30,000 units.

The intramuscular injection is usually the method employed to maintain the inhibitory titre of the blood. The deltoid or the gluteus muscles are the sites most frequently used. Since it is essential in the intramuscular injection to keep the volume as low as possible in order to avoid local discomfort at the injection site, the concentration employed is usually higher, namely about 5,000 units per cc. Taylor[4] recommends that this solution consist of physiological saline instead of distilled water, as this has been found to be less painful.

Local application may be made directly to the infected area and the solution used usually contains 250 units per cc. This method of local application is continued even when the other two methods of administration have been stopped.

The local application of the powdered sodium salt of penicillin has been found far too irritating for general use, as was noted by various investigators in England.

In view of the fact that the presence of penicillin was not demonstrated in the spinal fluid after intravenous or intramuscular injections, according to the investigations of Rammelkamp and Keefer,[1] it has become necessary to perform intrathecal injections for patients with meningitis. In certain cases Spurling[6] prefers the injection of penicillin into the lateral ventricles through a burr hole, rather than intraspinally, in order to insure better diffusion of the drug from above downward, but the intraspinal injection is also employed.

There have been certain untoward reactions encountered during the developmental period of penicillin therapy but the commercial products, as issued today, are satisfactory. Urticaria has been observed in a number of instances, and eosinophilia may occur; the highest

percentage reached of eosinophiles in our experience was 14 per cent and this receded to a normal eosinophile count 72 hours after discontinuance of the drug. These untoward reactions are believed to be due to the impurities present, which are not a part of the active penicillin but really constitute about 80 per cent of the product as used.

Penicillin is excreted in part by the kidneys and also to some degree through the bile.

Lyons[2] reports four cases of osteomyelitis of the skull, two cases of mastoiditis and one case of pansinusitis, all of which gave a most favorable response to the drug.

Postoperatively it is important to maintain a nitrogen balance; this may be accomplished by intravenous injection of either plasma, whole blood or amino acids. Just enough is given to maintain a positive nitrogen balance.

The actual dosage of penicillin cannot be outlined with any great precision. It varies from 90,000 units to 400,000 units daily, given in divided doses at three- or four-hour intervals, intramuscularly. However, staphylococcus requires ten times the amount of penicillin as does streptococcus. The index for this variation of treatment is dependent very largely on the clinical evidence of the response to the drug, but the agar plate culture with small wells formed in the agar plate give a definite index of the inhibitory titre present in the blood or the spinal fluid.

REPORT OF CASES

CASE 1. This patient had been previously treated surgically before the availability of penicillin.

This 28-year-old sergeant was admitted to the hospital on Dec. 5, 1941. The diagnosis was pansinusitis and osteomyelitis of frontal bone. He had received five successive operations for removal of sequestra of the frontal bone from the time of his admission until July 1942, a period of seven months. His case came under my observation after all of these other operations had been performed, and it appears that in spite of sulfonamide therapy and intranasal sinusotomy with apparent adequate drainage there appeared recurrent, progressive sequestrations of the frontal bone, requiring successive removal.

Two further operations were then performed externally, one on either side in the frontal area, whereby transfrontal ethmoidal revision was accomplished. A catheter was inserted on each side from

the frontal area into the nasal cavity and the external wound over the frontal area was completely closed.

Primary union occurred and the wound in the frontal area stayed healed for a period of about one month. Subsequently, a minute rupture occurred in the frontal area on each side, followed by a purulent drainage which, although not excessive in amount, was persistent and could not be cleared. Culture from the wound showed the presence of a staphylococcus aureus.

He was finally placed on penicillin therapy, whereby he was given a primary intravenous injection, followed by intramuscular injections every four hours for a period of seven days. At the same time local instillations of penicillin were given into both draining tracts in the frontal area. The drainage lessened after the first few days of treatment, and at the end of a week there was very scant discharge. He was then released from the hospital and appeared each day at the clinic for local instillations of the penicillin. After ten days of further instillations, the tracts were completely healed and there has been no discharge from these tracts since that date, a period of about three months. However, there is considerable deformity of the frontal bone due to the fact that practically the entire outer table had been removed, and a plastic repair is contemplated in the near future.

CASE 2. This 21-year-old seaman 3rd Class had an onset of severe headaches with chills and fever about Sept. 12, 1943. He had been placed on sulfonamide therapy.

He came under our care on Sept. 23, 1943, about two weeks after the onset. The previous history was not revelant to the present condition.

On admission he had a white blood count of 13,500; temperature of 99° F.; pulse 110; headache and profuse perspiration.

He presented a marked swelling of the right upper eyelid and the edema caused complete closure of the eye. An examination of the eye showed the eyegrounds to be normal. There was no proptosis of the eyeball and no interference with its movement. Intranasal examination revealed a very slight congestion of the nasal mucous membranes with no evidence of nasal discharge. There was some tenderness over the right frontal sinus.

An x-ray film of the accessory nasal sinuses revealed slight clouding of the right frontal sinus and a haziness of the remaining

sinuses on the same side. There was no roentgenologic evidence of osteomyelitis of the frontal bone.

The patient was placed on sulfadiazine therapy, constant steam inhalations and local shrinkage, and silver protein packs were inserted in the nasal cavity.

He showed immediate response to this therapy as evidenced by cessation of headaches, the almost complete resolution of the swelling of the right upper lid, and the loss of tenderness over the right frontal sinus within 48 hours.

On September 29, 1943, six days later, he developed a sudden pain in the right lower quadrant and back, with radiation of this pain to the groin, associated with dysuria and some hematuria. This was interpreted as a blockage of the kidney pelvis with sulfadiazine crystals. Sulfonamide therapy was immediately discontinued and intravenous glucose in sterile water was administered. The urinary symptoms promptly began to subside and two days later all urinary symptoms had disappeared and all signs and symptoms of the frontal sinusitis had become resolved. Three days following this, there was a recurrence of the swelling of the right upper lid with some tenderness over the right frontal sinus.

He was then placed on penicillin therapy, consisting of a primary intravenous injection, followed by intramuscular injections at four-hour intervals. His temperature was 102° F.; pulse, 120; respiration, 20. In 36 hours the swelling of the lid had become so tense that surgical intervention was decided upon. An x-ray examination at this time revealed no evidence of involvement of the frontal bone.

An incision was made through the right eyebrow and a subperiosteal abscess under great pressure was encountered. A dehiscence was found in the outer table of the frontal bone. The bone was rongeured around the area of the dehiscence. No attempt was made at this time to curette the sinus itself. A small catheter was inserted directly into the frontal sinus and sutured to the skin, and the sinus was then filled with penicillin solution. A culture taken showed no organisms and no growth. There was no drainage whatsoever from this wound following the final evacuation of the pus. Penicillin therapy was continued by intramuscular injections and by daily local instillation through the catheter. Following the incision the swelling immediately began to subside and continued to improve. Subsequent cultures revealed no organism. Treatment

was carried on until Oct. 14, when all parenteral penicillin was discontinued.

On Oct. 20, 15 days after the incision, the tube was withdrawn. The patient's general condition was excellent. He was up and about the ward and he had no complaints. Penicillin solution was still being instilled through the tract left by the tube, but since there was apparent resolution the wound was allowed to close. An x-ray film of the sinuses taken at this time showed a much improved illumination of the right frontal sinus.

Six days later the patient suddenly developed acute edema and hyperemia of the right upper eyelid, similar to his previous flare-up. There was a white blood count of 18,600 at this time and the temperature was 101° F. He was re-incised over the same site of the right eyebrow. Pus was again found under pressure and evacuated. The outer table of the right frontal bone was necrotic, with a small sequestrum at the area of the primary dehiscence. The sequestrum was removed and the periphery of this opening was then rongeured until healthy bone was obtained, producing a defect about 2 cm. in diameter. This time the diseased membrane of the frontal sinus was completely curetted and a considerable amount of granulations removed. A rubber catheter was inserted and the frontal sinus filled with penicillin. Thirty thousand units of penicillin were given intravenously, followed by twenty thousand units every three hours intramuscularly. A culture taken at this time revealed the presence of a gram-positive bacillus, which was identified as B. subtilis. The penicillin was continued until 1,000,000 units were given over a period of five days. Again no drainage whatever occurred following the incision and evacuation of the pus. The temperature came down to normal during this period, and the swelling progressively subsided. Daily instillations of penicillin were given through the catheter. The catheter was allowed to remain in place this time for three weeks. It was then removed and a small rubber dam inserted in its place for a period of two weeks, and then removed and the wound allowed to close. X-ray films showed no further evidence of bony involvement. Clinically, both subjectively and objectively, there was no evidence of any activity.

On Dec. 16 the patient was sent on a three-week furlough. He has just returned and to date there are no evidences whatever of any activity.

CASE 3. The patient is a 33-year-old sergeant, who has a long-standing sinus disease, consisting of intermittent headaches, nasal blockage and postnasal drip.

His present illness started on Nov. 3, 1943, when he developed an extensive swelling involving the forehead and running down the side of his face and closing both eyes. Because of the ballooning appearance, it was thought he had an insect bite. This swelling lasted only one day.

A swelling recurred three weeks later which lasted three days. A subsequent swelling recurred one month later, but this time it became localized over the center of his forehead. An x-ray film of the sinuses was taken, which revealed a pansinusitis. He was given local nasal treatment, but the swelling persisted without causing any edema about the eyes or face, and he was transferred to our care about nine days later.

On admission to our hospital the patient presented a circumscribed, tense, painful swelling 7 cm. in diameter in the middle of the forehead and involving the mesial 2 cm. of the eyebrows.

His temperature was 101.4; white blood count, 11,000. There were no meningeal signs. A diagnosis of osteomyelitis of the frontal bone was made. Operation was performed the following day. A vertical incision was made at the midline of the forehead, over a tense globular circumscribed swelling about 8 cm. in diameter, from the bridge of the nose upward for a distance of about 7 cm.

On incising the periosteum approximately 4 cc. of thick, grayish-yellow pus and a few air bubbles exuded. The periosteum was reflected laterally on either side bringing into view a dehiscence in the left frontal sinus near the midline, measuring about 1 cm. in length and about 10 mm. in width. About this opening was soft, spongy necrotic bone. This was rongeured away until firm, healthy bone was encountered, leaving a defect in the outer table about 2 cm. in diameter. The inner table was intact.

The septum between both frontal sinuses was soft and was removed, and polypoid membrane curetted from both frontal sinuses.

An 18 F. catheter was inserted into the sinuses and the wound closed with sutures. Penicillin, 250 units per cc., was instilled to fill the sinuses. A culture was taken and showed the presence of staphylococcus aureus.

An initial intravenous penicillin injection of 30,000 units was given and then 25,000 units of penicillin were given every three hours intramuscularly. When the patient had received 1,000,000 units, parenteral injections were stopped, but local instillations have been continued. The swelling and all symptoms have entirely sub-

sided, but the catheter is still in place. No further drainage has been noted and the temperature has remained normal since the operation. The patient was again seen two months later. The wound remained entirely healed with practically no deformity.

Williams and Nichols[5] report two cases of spreading osteomyelitis of the frontal bone treated with penicillin. One of these cases was in a child aged nine years and another in an adult aged 35 years. An epidural abscess, as well as a superiosteal abscess with dehiscence in the frontal bone, was found in the child. However, there was apparently no epidural abscess in the adult. They are of the opinion that an epidural abscess is the usual concomitant lesion found when a subperiosteal abscess is present.

Obviously, since the frontal sinuses had not yet reached full development in the child, there actually was no real outer and inner table of the frontal bone. This, it seems to us, would account for the extension to form an epidural abscess in that case. Such a complication has developed in long standing cases in adults, but in our cases here reported it did not occur.

CONCLUSIONS

1. The use of penicillin in this type of infection gives promise to preclude the radical surgical procedures ordinarily previously employed with the attendant deformity.

2. We cannot depend upon penicillin alone unless drainage is established surgically. Therefore, penicillin definitely has its limitations and must be used as a supplementary measure to surgery, or vice versa.

3. Drainage stops fairly rapidly after primary evacuation of the pus where penicillin therapy is instituted.

4. Constant or frequent local penicillin application is essential even after cessation of the parenteral injections.

5. At least wo weeks of serial, sterile cultures of the wound should be obtained before allowing a wound to close.

6. The fact that penicillin has overcome the acute infections caused by staphylococci and the sulfonamide-resistant streptococci indicates that penicillin is an exceptionally potent antibacterial agent.

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XLVII

STEREOSCOPIC VERSUS PLAIN FILMS IN ACCESSORY SINUS EXAMINATIONS

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Roentgenography of the nasal accessory sinuses is the most difficult of the many branches, and requires the most painstaking care in the technique. A poorly made set of films is worse than useless. There must be a sufficient number of films made in various angles, and it is astonishing how many times I have films referred for interpretation and find there are only two films. Such a set is of no value whatever.

There are three methods for this examination: (1) plain or flat films; (2) stereoscopic films; (3) sectional or planigraphic films.

All roentgenologists can make plain or stereoscopic films but only a few have access to the equipment needed for planigraphic films. The most generally satisfactory examination is with stereoscopic films. Very few rhinolaryngologists have their own stereoscope, but all hospitals and roentgenologists have the instrument. In case none is convenient, it is possible to so train the eyes that stereoscopic vision can be obtained without the use of any extra equipment. It is very simple and, once it is understood, requires little practice to obtain a good third dimensional impression.

Of course, there are certain individuals who cannot relax and fuse the two images, even with the best of mechanical apparatus. In this case he still has the conventional plain film to look at, but the stereoscopic pair is available, if necessary.

The planigraph is a wonderful achievement and in a few cases is valuable in sinus work, but it requires special training in the technique and the interpretation. It is indispensable in the examination of the larynx in the posterior anterior position as it does remove the image of the spine and shows the extent of involvement of the soft tissues of the larynx.

Read before the Sixty-Sixth Annual Meeting of the American Laryngological Association, New York, N. Y., June 7-8, 1944.

Why are not more stereoscopic examinations made? There are several reasons: the additional expense to the roentgenologist; his inability to collect a larger fee to compensate; the lack of training of the technician; the roentgenologist's inability to fuse the images and appreciate the added value of the third dimension on a film.

When you examine a disarticulated skull you see depth, so why not see depth in the x-ray film? It is a great help and once you realize the added information you will insist upon stereoscopic films for all your cases. It is easier to visualize pathology when the film depicts the condition as it would appear if you were looking into the sinus rather than at it.

When will sterescopic films give added information?

1. In all cases of fracture of the skull.

2. In all cases of fracture of the nasal bones. They enable one to see which side is fractured and how much displacement exists.

3. In cases in which the film taken in the Waters position shows an apparent tumor in the antrum. A stereoscopic film may show this tumor to be due to the image of the cheek or upper lip superimposed on the antrum.

4. In cases in which the film taken in the Waters position is negative, while the film taken in the 23° angle position is positive. A stereoscopic film may show this opacity to be due to an increased density of the floor of the posterior cranial fossa, which will be placed posterior to the antrum.

5. In cases in which it is difficult to determine whether the right or left ethmoid shows a change in the appearance of the cell structure as viewed on the lateral film.

6. In cases in which it is difficult to determine on which side a large agger cell exists.

7. In all cases of enlargement of the sella turcica.

8. In all cases of bony involvement of the skull.

9. In cases in which there is a suspicion of absence of a frontal sinus or a four plus involvement of a frontal as indicated by a faint circular line of density where the superior border of the frontal should be. A stereoscopic film may show this line to be part of the occipital suture superimposed on the frontal region, and not the superior border of the frontal, thus proving the absence of a frontal sinus.

10. In cases in which the lateral film shows a suspicion of a partition dividing the antrum into two sections. A stereoscopic film will prove this apparent partition to be the posterior border of the malar bone.

11. In all cases for examination of the petrous pyramid.

12. In all cases for examination for salivary calculus.

13. In cases in which an apparent soft tissue tumor is shown on lateral films of the pharynx. A stereoscopic film may show this to be caused by the lobe of the ear and to be entirely outside the skull, not in the pharynx.

There is one precaution to be observed when examining each one of a stereoscopic pair of posterior anterior views as separate plain films. The necessary shift in the position of the tube may cause one antrum to be more opaque than the other. This opacity may be manifest in both views but on opposite sides. The stereoscope will fuse them both and give you a true image. If you do not use a stereoscope, then consider the condition of both antra to be as registered by the lesser opacity. The increased density is caused by the angulation of the tube and shifts as the tube is modified to one side or the other. Of course, if one or both antra are badly involved, this precaution does not follow.

14. In cases of malignancy involving the antra. The stereoscopic film enables one to determine more accurately the degree of bony involvement.

From the above, it is evident that stereoscopic views are not absolutely essential in all cases but the roentgenologist does not know the clinical evidence and it is embarrassing to have to request a second examination to clear up some disputed point.

Many of you send your patient to the roentgenologist with a request for a sinus examination without giving any information about the case. Some patients object to questioning by the roentgenologist and thus he may be handicapped by lack of information. A brief note, or phone call, would greatly facilitate the technique and aid in the interpretation.

Perhaps you are saying I am bigoted and have a special desire for stereoscopic films but I find them of great assistance, and am sure you would gain more information if you could succeed in the tech-

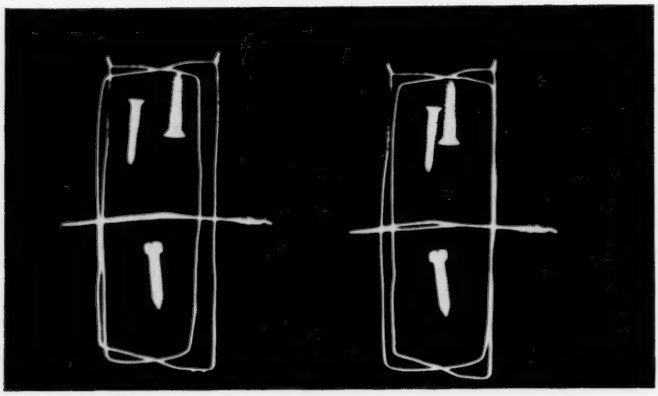

Fig. 1

nique of obtaining stereoscopic vision without the use of expensive and bulky apparatus.

I have given you the reasons for wishing stereoscopic films; now I will try to demonstrate to you the method of obtaining third dimensional impression. Fig. 1 shows a pair of stereoscopic films of a card board box, bound with wire, and suspended within are three screws at different levels. The idea is for you to converge your eyes so that there will be crossed vision. You should see the left hand image with the right eye and vice versa. If you were viewing two films in the illuminating box, one alongside the other, you should step back three or four feet and follow my directions.

Place the open hands against the side of the orbits, folding the thumb in. Keep the hands straight, like blinders on a horse. Close the left eye and swing the right hand over like a gate, so that the image of the right hand film is obscured. Keep the hands there. Open the left eye and close the right eye. Swing the left hand over until the image of the left hand film is obscured. Keep both hands steady. Now open both eyes. If you see two images try to focus on one or the other, try to pull them together. You should see everything in the third dimension, the wires forming a square and the screws suspended within the square. If unsuccessful, start all over again. You must relax and converge.

After you have mastered the method you can dispense with your hands and converge the eyes at will and see stereoscopically. Thus

you could have a pair of stereoscopic films in the operating room and simply look up at them and see the part you are operating upon with the same sensation of depth as the actual structure on the table.

The best films to practice on are chest films and those of an empty skull. Many of you may never have seen a stereoscopic image and not understand what it means. If you have available an old-fashioned parlor hand stereoscope and some pictures, you will understand what that impression of a third dimension really means.

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XLVIII

VENTRICULAR LARYNGOCELE

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It was in 300 A. D. that Galen[1] first made mention of the laryngeal ventricle. Recognition of this recess was credited to him by Morgagni,[2] who gave an incomplete description of it in 1741 and for whom it is named. Mackenzie[3] advocated calling it the ventricle of Galen. Eighty-eight years after Morgagni's account was published, Larrey, the French army surgeon, first mentioned the air sac which at times develops from the saccus of this ventricle. The term laryngocele, now commonly applied to this anomaly, was introduced in 1867 by Virchow,[4] who described the sac in these words:

> "I have found a dilatation of the ventricle of Morgagni, up to the present unknown as it seems, which one may call 'ventricular laryngocele.' One sees, in these cases, small elongated sacs with thin walls, coming off from the upper part of the ventricle by a somewhat narrow orifice, and sometimes reaching to the upper border of the thyroid cartilage, and even to the hyoid bone, where they terminate by an end in the form of a club ; most frequently they are found only on one side; once, however, I have met them on both sides. Their walls are smooth, their interior is lined with ciliated epithelium, and they are filled with air."

Confusion in nomenclature makes questionable the number of cases of laryngocele reported up to the present time, but at best it is small. Hipple,[5] in 1910, reviewed the world literature on the subject of pathologic distension of the appendix of the ventricle into an air-containing cyst and found only 20 cases reported. In but 12 of these cases was the condition observed in life. Three years later, Hansberg[6] stated that 24 cases had been published up to that time. In 1921, Iglauer[7] reported a case. Moore,[8] whose comprehensive article was published in 1922, has been misquoted in regard to collecting 85 cases of laryngocele in the literature. The confusion arose because his article was on the complete subject of "The So-Called Prolapse of the Laryngeal Ventricle and Eversion of the Sacculus" and was not confined to a discussion of laryngoceles. He merely cited 10 cases as examples of ventricular laryngocele as described by Virchow.[4]

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In the recent American literature, Jackson and Jackson[9] in their textbook published in 1937 mentioned two cases. Other authors reporting cases are Freedman,[10] two cases and Simpson,[11] one case in 1938; Lindsay,[12] one case in 1940; Allman and Cordray,[13] two cases in 1942; Lothrop,[14] one case in 1943; and Schall,[15] one case in 1944. In Schall's case the laryngocele was associated with carcinoma of the larynx; the author believed it to be the first case of the kind reported in the literature. It is of interest that during the 66 years of the existence of the American Laryngological Association there appears but one report of a case of laryngocele in the Transactions of that senior laryngological society; in 1914, Shambaugh[16] presented a case in a paper entitled "Laryngocele Ventricularis."

The proper clinical conception of laryngocele in man is dependent in large measure upon adequate understanding of the evolution and physiologic aspects of the ventricle of Morgagni. This portion of the larynx is a horizontal space, elliptical in shape. It is situated between the true and the false vocal cords. Anteriorly from the roof there arises a vertical pouch known anatomically as the sacculus. Moore,[8] citing Rudinger,[17] stated that the ventricles are relatively much larger in the male and added that occasionally they are greatly overdeveloped.

There is a close resemblance between the larynx of mammals and that of reptiles and birds. One striking difference, however, is that the ventricle is observed only in mammalian life. The human larynx was not originally designed to serve as an organ of speech, but instead was primarily intended as a valve for guarding the pulmonary system at its entrance by exercising the primitive function of keeping everything out of the lungs but air. Negus[18] concluded that air sacs are not developed for purposes of voice and noted that many animals without them are as noisy or more so than those with them. Among the examples he cited are the roar of the lion, which has no air sacs, and the seldom heard voice of the Anubis baboon with its enormous air cavities; also, the howler monkey with thin osseous air sacs is especially noisy, and the lemur without these sacs is likewise extremely noisy.

As viewed by this author, the structure of the larynx in the different mammals differs in accordance with the relative quantity of oxygen needed and of carbon dioxide given off by the lungs. This requirement is influenced by the mode of life. In anthropoid apes, for example, temporary cessation of respiration is involved in the fixation of the bony structure of the chest required to give the pectoral muscles full play as the forelimbs grasp, hug, strike and climb.

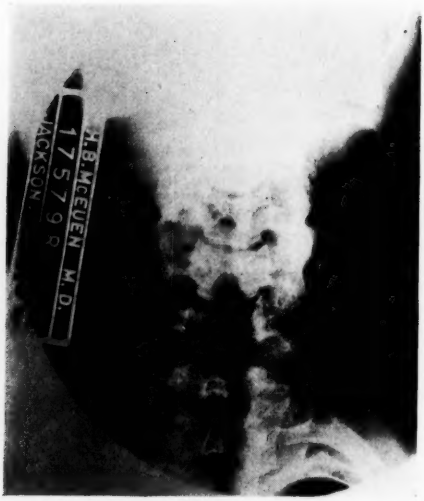

Fig. 1.—Anterior posterior view of air sac.

Compensation for the interruption of the vital function of normal respiration was attained by the evolution of air sacs arising from the ventricle of the larynx. Filled with oxygen, and during expiration with carbon dioxide, they serve as mixing chambers and are used with the least amount of exertion by the lungs.[12]

It would appear that the sacculus, which is a constant finding in man, is a degenerated relic of air sacs leading out of the ventricle, or perhaps their incomplete precursors, with a function directed to rebreathing air.[12, 18] It contains lymphoid tissue and mucous glands, which without doubt help to supply the continuous flow of mucus required to keep the borders of the vocal cords lubricated during phonation. This recess varies in size from a simple depression near the anterior end of the ventricle to a narrow prolongation extending up to the level of the thyroid cartilage and, rarely, even higher. Comparison of the human sacculus with the excessively large air sac of the chimpanzee or the orangutan, as Negus[18] noted, clearly indicates evolutionary developmental changes degenerative in character. It is from this appendix that the ventricular laryngocele develops.

Lindsay[12] described three types of laryngocele. The internal laryngocele, commonly observed, is a cystic dilatation in which the

Fig. 2.—Lateral view of air sac.

hernial protrusion of the mucous membrane lies within the larynx. The superior external laryngocele, occurring less frequently, appears as a swelling in the neck after the sac has perforated and projects as a hernia through the thyrohyoid membrane. Also, the internal and external laryngocele may occur in combination, joined by a small isthmus. In this mixed type the extralaryngeal portion is frequently large and has usually protruded through the thyrohyoid membrane.

A preexisting pathologic condition is not necessary to the development of a laryngocele for the condition is observed in the normal larynx. Etiologic factors mentioned in the literature include coughing, whooping cough, singing, horn blowing, glass blowing, weight lifting, vomiting, straining at stool and childbirth. The known occurrence of a congenitally long appendix of the ventricle may also be of etiologic significance.[12] In all cases there is present an abnormally increased intraglottic pressure, to which the ventricles and, in turn, their appendices are exposed. Too, it is probable that tumors, syphilis, tuberculosis and other diseases affecting the larynx may so weaken the walls of this organ that a laryngocele may arise, especially in the presence of other etiologic factors.

If the mucosal hernia is small, there are no symptoms. The size of the air sac may, however, increase to the extent that it causes em-

barrassed respiration and even dyspnea, and it may also affect the normal function of the vocal mechanism. The internal laryngocele, which sometimes extends to the aryepiglottic fold or even to the base of the tongue, dilates on forced expiration and, if large, may produce some obstruction accompanied by hoarseness.

When the sac has perforated through the thyrohyoid membrane and appears as a swelling in the neck, coughing, crying, physical exertion with the upper extremities, or straining at stool may cause the superior external laryngocele to become enlarged. It can be emptied by pressure, and the sound of escaping air, or air and fluid if infection is present, may be heard. Local discomfort and headache may accompany the distention if there is pressure on the large veins of the neck. When a laryngocele becomes infected and a pyocele develops, the patient on coughing may expectorate pus.

An external swelling of the neck with convexity of the ventricular band, some decrease of this mass on pressure accompanied by a hissing sound, and the presence of a tympanitic note on percussion should establish a clinical diagnosis, which may readily be confirmed by roentgenograms showing a sac filled with air. In the earlier cases, when a laryngocele was suspected the introduction of an aspirating needle to determine the contents of the tumor-like swelling was a diagnostic measure. This procedure which might introduce infection, became unnecessary with the advent of the roentgenogram. Roentgen studies now make easier the diagnosis of a laryngocele and offer but another evidence of the fact that no laryngeal examination is complete without a roentgen study.

REPORT OF A CASE

J. C. F., a white man aged 35, was referred to me with a tentative diagnosis of carcinoma of the larynx. He could not recall when he had not had a recurring hoarseness and a swelling on the left side of the neck. He related that since childhood he had been fond of singing and possessed a voice of considerable volume. Oftentimes, on singing, he had noticed that the swelling would increase in size; also, the forced expiration associated with muscular exertion, such as the lifting of a weight and particularly straining at stool, would cause the mass to become larger. During the two years immediately preceding the examination, he had noticed enlargement of the swelling, increased hoarseness and occasionally embarrassment on forced inspiration. At times, on pressure over the mass he had observed that quick reduction in its size was accompanied by a squeaking or hissing sound.

Local examination disclosed on the left side of the neck the presence of a tumor-like swelling about the size of a tangerine, which extended above the thyroid cartilage and increased in size on coughing. The patient voluntarily percussed this external mass, which gave forth a tympanitic sound. Indirect laryngoscopy showed a swelling involving the left ventricular band, which at its greatest convexity projected beyond the median line of the glottis occluding entirely the view of the left vocal cord. A roentgen study established the diagnosis of ventricular laryngocele.

On July 10, 1942, with the cooperation of Dr. Kenneth Morris, the tumor was successfully removed without rupture. An incision was made on the left side of the neck at the level of the thyroid cartilage, extending from the middle of the sternomastoid muscle forward to about an inch from the midline. When the platysma muscle was cut and the superficial fascia opened, a thin-walled sac protruded between the sternohyoid muscle medially and the sternomastoid muscle posteriorly. It was in rather intimate contact with the submaxillary and lingual vessels above and the carotid vessels laterally. The sac was dissected free down to the pharynx where the neck could be seen coming from the posterior lateral aspect of the pharynx just above the thyroid cartilage. The neck of the sac was doubly ligated with crochet cotton and cut between the ligatures. The sac was removed intact. The stump was enfolded with several cotton sutures and muscle tissue, and fascia was used to cover the stump. A small rubber dam drain was inserted to the deepest portion of the wound, and a small amount of sulfanilamide was sprinkled around the stump of the sac. The wound was closed around the dam with interrupted cotton sutures.

The pathologic report, made by Dr. L. Y. Dyrenforth, follows:

"*Gross*: This mass is about 5 cm. in diameter and is open at one side by a small neck of wrinkled and edematous tissue, giving it somewhat the appearance of the closed part of a toy balloon. The wall is uniformly about 2 mm. in thickness, but at the opening the tissues are edematous and consequently thicker than this. The interior is fairly smooth, but near the opening there is distinct rugation, which becomes more pronounced as the aperture is approached. The interior surface is translucent gray, with a distinctly mucosal aspect. The external surface is slightly ragged. A quantity of limpid serous fluid is present in the unopened specimen, which does not competely submerge in a beaker of ordinary 10 per cent formalin.

"*Microscopic*: The wall of this sac is characterized by a uniform consistent inflammatory reaction. This reaction manifests itself by clumps of round cells and other mononuclears of inflammatory origin. These occur mostly in multiple foci and are sprinkled through the adventitia. There are considerable numbers of well developed lymphoid follicles also in the loose tissue beneath the mucous membrane. These contain well developed follicle formations and germinal centers. Capillary

blood vessels are abundant. There are definite areas of granulation tissue near the surface. The mucous membrane is characterized by a high columnar epithelium suggestive of ciliated variety. There is an abundant inflammatory reaction beneath this. The deeper layers of the stroma contain muscle fibers and loose fatty tissue (Fig. 6).

"*Diagnosis*: Laryngeal cyst showing inflammatory changes in the wall."

The patient had an uneventful recovery. The drain was removed on the third day, and the sutures were removed on the seventh day. Healing was completed at the end of two weeks. The wound was examined again after three months had elapsed; it was well healed with no evidence of the laryngocele. It was noted at this time that the patient's voice which had previously been hoarse, was now practically normal in tone. Approximately two years later, on May 22, 1944, the patient was again examined. There was no evidence of recurrence of the cyst. Both the larynx and the voice had returned to normal.

SUMMARY

A survey of the subject of laryngocele in man including its phylogenetic aspects is presented.

One case is reported, in which the air sac was successfully removed intact. After approximately two years there was no evidence of recurrence.

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XLIX

INTER-AMERICAN RELATIONS IN OTOLARYNGOLOGY A REPORT

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During the past few years, many of our medical societies have been starting to direct attention to the promotion of better relations and understanding between physicians and surgeons of this country and those of the other American republics. This has been in part due to the Good Neighbor Policy of our government, expressed through both the State Department and the Office of the Coordinator of Inter-American Affairs. Even without these influences, however, such a movement is a natural consequence of the disruption of the previous relations of all the Americas with Europe.

I shall take the liberty, in this paper, of quoting and paraphrasing passages from a report prepared for the American Academy of Ophthalmology and Otolaryngology.[1] In this report I sketched briefly the history of international relations in otolaryngology as shown by the succession of international congresses that have been held since the First International Otological Congress, which took place in the United States, under the presidency of Dr. John Roosa about 70 years ago. The next one was held in Milan, and subsequent congresses met at Bâle, Brussels, Florence, London, Bordeaux and Budapest; the Ninth, the last before the outbreak of World War I, was held in Boston in 1912, with Clarence Blake as president.

The war caused a hiatus of ten years, but the Tenth International Congress of Otology was held in Paris in 1922 under the presidency of Professor Sébileau. This congress was not truly "international", because representatives of Germany and Austria were not invited. Six years later a Congress was held in Copenhagen under the presidency of Professor Schmiegelow, which it was decided to call the First International Congress of Oto-Rhino-Laryngology, instead of the Eleventh International Otological Congress. This Congress was a successor to the ten Otological Congresses, and three "Laryngo-Rhinological" Congresses. It seemed as if the succession

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of International Congresses was to be permanently resumed. A fine Congress of Oto-Rhino-Laryngology (the Second) was held in Madrid in 1932, with Professor Tapia as president, and the Third International Congress of Oto-Rhino-Laryngology took place in Berlin in 1936 under the presidency of Professor Carl Von Eiken. This was a great success, and a well balanced representation of otorhinolaryngologists from many nations of the world participated. The British, French and Belgian delegations were especially numerous, which seemed a sign pointing toward the development of real friendship and collaboration. In Berlin it was voted that the next Congress of Oto-Rhino-Laryngology (the Fourth) should take place in Amsterdam in 1940, under the presidency of Professor Burger. Plans for this congress were being energetically prosecuted until the spread of World War II made it obvious that cancellation would be necessary.

Let us outline briefly some aspects of the history of otolaryngology on the South American continent. According to Dr. Aristides Moll, Secretary of the Pan-American Sanitary Bureau, otolaryngology was "among the first of the specialties to forge ahead, usually tagging along close behind her elder sister, ophthalmology"! Dr. Carlos Desverine of Havana was one of the first Latin Americans to devote himself to the specialty (1887), and Dr. Conde Flores of Venezuela was another pioneer (1895). Institutions for the deaf were established in Rio in 1856, Buenos Aires in 1857, Mexico in 1869 and Havana in 1925.

In 1936 a historic meeting was held in São Paulo, Brazil, under the presidency of Dr. Mario Ottoni Rezende, which was called the Paulista Otorhinolaryngological Week. At this meeting it was decided to hold a First Brazilian Congress of Oto-Rhino-Laryngology, after an interval of two years. This First Brazilian Congress took place in 1938, in Rio de Janeiro, with Professor Marinho as president, and at its conclusion a decision was reached to join with colleagues of the other nations of South America in a First South American Congress of Otorhinolaryngology. This First South American Congress of Otorhinolaryngology was held in Buenos Aires in 1940, under the presidency of the dean of Latin American otolaryngologists, Prof. Eliseo Segura and at its conclusion a First Latin American Congress was planned, to be held in São Paulo, Brazil, in 1943, under the presidency of Professor Paula Santos. However, since it was deemed impracticable to hold a Congress at this time in São Paulo, it was decided to have, instead, a Second South American Congress in Montevideo, with Professor Justo Alonso as president. This Congress is to

take place in November of this year (1944). In the light of this historical background, there can scarcely be any doubt of the advisability of taking steps toward the organization of a Pan American Congress of Otolaryngology.

Thus far, the only occasions on which the otolaryngologists of the United States have met in scientific session with those of the other American republics have been during the Congresses of the Pan American Medical Association. At the Fourth Congress of this Association held in Dallas, Texas, in 1933, Dr. Chevalier Jackson Sr. was president of the Section on Otolaryngology; he was again president of the section the following year for the Fifth Congress, which was a "floating" congress stopping at Havana, Panama, Caracas and San Juan. The Sixth Congress was held in Rio in 1935 and the Section was presided over by Dr. Lee Hurd. Finally, the Seventh Congress of the Pan American Medical Association took place in Havana in 1938, and Dr. William E. Sauer presided. The essayist had the honor of being a sort of "perennial" secretary of the Section in all of these congresses.

On January 19, 1942, Dr. Wherry wrote me that the Program Committee of the Academy had appointed me as chairman of a committee to develop plans for a Pan American Congress of Otolaryngology. Because of the increasing difficulties in communications and transportation resulting from our progressively more complete involvement in the war effort, nothing was done during the ensuing months except to write informally to some of our Latin American correspondents concerning the project of a Pan American Congress. Throughout the spring months of 1943, Dr. Babbitt, then president of the Academy, sent letters to a number of representative otolaryngologists in the various other American republics, telling them about the meeting of the Academy in Chicago in October 1943 and inviting them to attend. Despite the great difficulties in communication and transportation due to chaotic international conditions imposed by war, a very gratifying response rewarded our efforts. A number of distinguished Latin American otolaryngologists made the trip to Chicago, and a number of others accepted the invitation to attend, but were prevented from reaching Chicago by circumstances beyond their control. The great majority of those who did not accept wrote us expressing their appreciation of the invitation and their warm sympathy with the idea of establishing some sort of Pan American organization in otolaryngology.

The entire meeting of the Academy was pervaded with the spirit of inter-American friendship and cooperation. It was very appro-

priately keynoted by the magnificent address of the Guest of Honor, Dr. Ralph Fenton, on "The Unity of Medical Science in the Americas" on Sunday afternoon. At the Inter-American Dinner on Wednesday night, President Babbitt welcomed the Latin American guests in Spanish, and they were then introduced individually. Addresses of the evening were given by Mr. William L. Schurz of the Division of Cultural Relations of the State Department, and Colonel Albert Dreisbach, of the Office of the Coodinator of Inter-American Affairs.

Our Committee on Inter-American Relations met for the first time at breakfast on Sunday morning, October 10. At this meeting Dr. Gradle was asked to speak with regard to the project of a Pan American Congress of Otolaryngology, from his experience in connection with the establishment of the Pan American Congress of Ophthalmology. He outlined briefly the story of the ophthalmological organization and made numerous suggestions regarding the proposed otolaryngological one. Various members of the Committee, and the Latin American colleagues present discussed the project of a Pan American Congress of Otolaryngology. Several of the members of our Committee expressed the opinion that there would be numerous advantages in holding the first Pan American Congress in conjunction with, or immediately following an annual meeting of the American Academy, as the First Pan American Congress of Ophthalmology had been held. Subsequent congresses would be held in the other American republics, perhaps in conjunction with the meeting of one or another of the Latin American societies.

At the conclusion of the meeting it was moved by Dr. Fenton that the Academy be requested to sponsor the establishment of a Pan American Congress of Otolaryngology and to finance its initial meeting, this initial meeting to be held in conjunction with a meeting of the Academy. This motion was seconded by Dr. Theobald, and passed. Two additional meetings of the Committee were held before the close of the Academy sessions, and the Committee was informed that the Council of the Academy had agreed to sponsor a Pan American Congress of Otolaryngology to take place in connection with the annual meeting of the Academy in 1945.

The Academy Committee is continuing as both a Committee on Inter-American Relations in Otolaryngology and a Committee on Pan American Congress of Otolaryngology. The following is a complete list of the members of the Committee as it now stands: *Executive Committee*: Chevalier L. Jackson, chairman, James A. Babbitt, Ralph Fenton, Juan Font, Arthur Proetz, Frank Spencer, Gordon B. New. *Members*: Victor Alfaro, Howard C. Ballenger, Wesley Bow-

ers, Mackenzie Brown, Thomas Carmody, George M. Coates, William Gill, Perry Goldsmith, Paul Holinger, Charles J. Imperatori, Francis Lejeune, James M. Robb, William E. Sauer, LeRoy Schall, Marshall Taylor, Walter Theobald. *Consultants*: Harris P. Mosher, Moacyr Alvaro, Conrad Berens, Harry Gradle, Forrest J. Pinkerton. *Ex-Officio*: Lawrence T. Post, President, William L. Benedict, Executive Secretary.

During the current year this committee is keeping in touch with all of the Latin American colleagues with whom contact has been established, and establishing contact with additional ones. Letters are being written to the various societies of otolaryngology in the other American republics, acquainting them with the intention of the American Academy to sponsor a Pan American Congress in 1945, and extending to them an invitation to join with the Academy in organizing it. The Mexican Society of Ophthalmology and Otolaryngology already assures us of its fullest cooperation. The Cuban Society has pledged its support through its President, Dr. Pedro Hernandez Gonzalo. The support of the Colombian Society of Ophthalmology and Otorhinolaryngology is pledged by its President, Dr. Jorge Suarez-Hoyos.

Professor Justo Alonso, President of the Second South American Congress, has been informed of the decision of the American Academy to sponsor the establishment of a Pan American Congress of Otolaryngology and finance its initial meeting, to be held in conjunction with the Annual Meeting of the Academy, in 1945. Professor Alonso has already indicated his approval of the idea of organizing a Pan American Congress, and he has expressed his willingness to receive as delegates to the Second South American Congress this November members of the American Academy whose names we send him. He has already been sent the names of the members of our committee, and we should be glad to send on the names of any other otolaryngologists who are interested.

Preliminary steps are being taken to carry out the suggestion of Dr. Paul Holinger, that we endeavor to "promote the exchange of post-graduate students and residents in otolaryngology between the United States and the other American republics." A form letter is being prepared, to be sent to all of the institutions offering approved residencies in otolaryngology, asking them what opportunities they have to offer for Latin American postgraduate students and residents. One man, Dr. Victorino Marquez of Venezuela, has already been given a residency at the Manhattan Eye and Ear Hospital through the efforts of our Committee.

In conclusion, I may say that in organizing the Pan American Congress for 1945 the Academy Committee will look for moral support and wise counsel to the American Laryngological Association. Needless to say, the Association will at all times be kept officially informed regarding plans for the Pan American Congress, since its secretary, and its present president, are members of the Academy Committee. I hope that our new president will consent to become a member of this committe also. Finally, I take the liberty of suggesting that some representative Latin American otolaryngologists be made Honorary or Corresponding Fellows of this Association. We have, in the past, had two distinguished Cuban otolaryngologists as Corresponding Fellows, Dr. Carlos Maria Desverine y Galdos, and Dr. Enrique Fernandez Soto, but at the present time we have no Latin American members.

255 SOUTH 17TH STREET.

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Argentina:

Sociedad de Otorrinolaringología de Buenos Aires,

Sociedad Rioplatense de Otorrinolaringología, Buenos Aires.

Sociedad Argentina de Otorrinolaringología de la Asociación Médica Argentina.

Sociedad Argentina de Bronco-esofagología.

Sociedad de Otorinolaringología y Broncoesofagoscopía de Cordoba.

Brazil:

Sociedade Oto-Rino-Laringologia de Rio de Janeiro.

Sociedade de Oftalmologia e Oto-Rino-Laringologia de Rio Grande do Sul, (Porto Alegre).

Sociedade de Oftalmologia e Oto-Rino-Laringologia da Baía.

Sección de Otorrinolaringologia de la Associacão Paulista de Medicina.

Chile:

Sociedad Chilena de Otorrinolaringología, (Santiago).

Colombia:

Sociedad Colombiana Oftalmológica y Otorrinolaringológica, (Bogotá).

Cuba:

Sociedad Cubana de Oto-Laringología.

Mexico:

Sociedad Mexicana de Oftalmología y Otorrinolaringología.

Paraguay:

Sociedad de Oftalmología y Otorrinolaringología del Paraguay, (Asunción).

Uruguay:

Sociedad de Oto-Rino-Laringología del Uruguay.

Venezuela:

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Venezuela:

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Aptdo. 1428, Caracas.

L

NASAL INJURY IN DYSTOCIA

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The study of the early stages of evolution of the human face gives us a general idea of the complexity of the factors that enter into the moulding of the individual nose.

Gregory[1] states that the transitional stages in the evolution of the nose from fish to man are fairly well understood and that the resemblances of the shark nose to the human nose are fundamental, and that the subsequent changes in this organ are not relatively great.

The human nose, both externally and internally, has precisely the same parts as that of the gorilla and the chimpanzee differing only in the matter of proportions.

The construction of the human nose begins in fetal life. In the embryo of 35 days, the nose is broad and flat with the nostrils set far apart and directed forward. In later fetal life, the formation of the nasal sacs takes place. Soon the primitive palate comes into existence from the proliferation of the mesenchyme beneath the floor of the nasal sacs. The nasal fossae grow dorsally until they meet the ectoderm of the oval cavity posteriorly, and the choanae become established by the disintegration of the epithelium at the meeting point of the nasal and oral layers. The olfactory sacs then extend into a more ventral position until they approach the midplane. The two nasal sacs, crowding to get to the center of the primitive face, compress the region between them. The septum thus formed from the fusion of the mesial nasal processes is subjected to pressure laterally from the nasal sacs, and caudally from the growing palate. The septum thus becomes subject to asymmetry, spurs and ridges and sublation of the anterior cartilage. During the late fetal months, the bridge of the nose becomes elevated and the nostrils begin to point downward. The degree of elevation of the bridge, however, is not

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From the department of Otology and Rhinology, Temple University School of Medicine.

Presented before the American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc., at the New York Academy of Medicine, New York, N. Y., February 25, 1944.

the same in all individuals and the dorsum may be straight, convex, or curving forward in accordance with the various nasal types.

During parturition[2] many nasal injuries result because of dystocia. Time will not permit discussion in detail of the various forms of abnormal labor such as dry labor, pelvic deformities, various presentations and positions all of which are added factors predisposing to nasal injury.

Labor should be a normal function of the human female. Any deviation from normal in the proportions of the infant or in the pelvis, or any interference with normal physiologic labor may result in dystocia.

There are comparatively few eutocias or absolutely normal labors, and it is difficult to draw the line where eutocia ends and dystocia begins. Any enlargement of a part of the infant in its relationship to the pelvic outlet will give rise to dystocia.

The increased size of the child's cranium is due to the increased mental capacity of the human race; and the greater development of the frontal lobe and its position create a larger cranium and may cause injury to a small part of the infant's body, such as the face or nose.

Many biologists theorize that, with the development of the nose and the septum, the septum has become buccaled because the space now occupied by the frontal lobe has prevented the septum from pushing upward and it is thereby deflected.

Metzenbaum[3] states that,

> "During a normal birth the skull conforms to the diameter of the osseous pelvis. This is accomplished through the compressibility given to the skull by the anterior and posterior fontanels and through the approaching and overlapping of the cranial bones. The tip of the lower half of the nose which protrudes accommodates itself to the pelvic diameters because it is flexible and easily depressed."

The processes of conception, development and parturition have many phases and most of these are very well understood. Academically, we are concerned with the following factors:

The adnexa uteri of the mother which rise into the abdominal cavity and descend into the pelvis.

The embryo, an aquatic being in its early stages, has at the end of the third month developed into the fetus; and at the end of the full term, must find his way through the tunnel to reach the outside world.

Consideration must be given to the dimensions of the skull and body which must pass through the cervix uteri, pelvis and perineum.

The motive power furnished in the physiology of childbirth comes from the uterus, the abdominal muscles, hormones, chest, closure of the glottis, diaphragm, and the hydraulic pressure of the amniotic fluid. The uterine contractions gradually increase and at maximum activity create a pressure of 30 to 40 pounds.

Imagine the abuse the nose must take with all that pressure behind it in abnormal labor!

Many obstetricians have experienced the sensation of uterine pressure upon their hands while the uterus is contracting. This is so great that it causes temporary paresis or complete anesthesia. Dealing with such a force, one can readily appreciate its damaging effect upon many parts of the body of the infant whether the delivery be normal or abnormal.

In classical normal delivery, both legs of the infant are crossed and flexed upon the thighs; the thighs rest upon the abdomen; the arms are folded across the chest and the head is flexed on the sternum. Frequently, however, the tibia and the fibula are bent due to the pressure exerted by the uterus during labor and many gross injuries result, such as caputsuccedaneum, brain hemorrhages, anoxemia, paralysis of the limbs, ptosis and injury of the nose.

The obstetrician concerns himself with the proportions of the pelvis to the most prominent fixed parts of the head and the relationship of the head to the outlet. Any minimal disproportion of either of these parts may convert a eutocia or normal labor into a dystocia or abnormal labor.

The classical measurements of the head within the normal limits have been given in our literature as follows:

Suboccipito-bregmatis (S.O.B.) 9.5 cm.

Occipito-frontal (O.F.) 11 cm.

Fronto-mental (F.M.) 9 cm.

Occipito-mental (O.M.) 13 cm.

The literature, however, makes no mention of the measurement of the occipito-nasal.

The human nose is exposed and protrudes anterior to the frontal bone and overhangs the superior and inferior maxilla. It seems

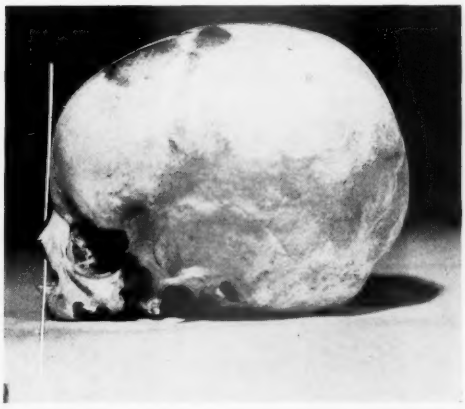

Fig. 1.—Fetal skull at birth with nasal osseous protrusion.

rather strange that no textbook nor any of our contemporaries have taken into consideration the bony protrusion of the nose in its relationship to the pelvic outlet (Fig. 1). Beside the bony protrusion, there is also the lobule of the nose or the cartilaginous part of the nose which is flaccid and compressible. No one seems to be cognizant of the soft protruding part of the nose in its relationship to the delivery and its implications. The soft protruding part of the nose includes the upper and lower cartilages and the quadrangular cartilage. The quadrangular cartilage articulates into the vomerian groove which is 1 to 1.5 mm. in width. In the classical measurements of the head, it appears that the nose has been completely overlooked.

During delivery various parts of the head become depressed causing a temporary obliteration of the fontanels and an overlapping of the cranial bones at the suture lines. We may presume that the reason the obstetrician has failed to take into consideration the protrusion or extension of the nose has been because he felt that it was soft, compressible, and dislocatable in character and that it would adapt itself to the external or surrounding pressure. The colloquial expression, "taking it on the chin" can very aptly be applied to "taking it on the nose" in this instance.

With the aid of Dr. N. Kendall, of Temple University Hospital, 25 newly born infants were measured using the comparative measurements of the occipito-frontal and the occipito-mental as the criteria in relationship to the occipito-nasal. To our great amazement it was

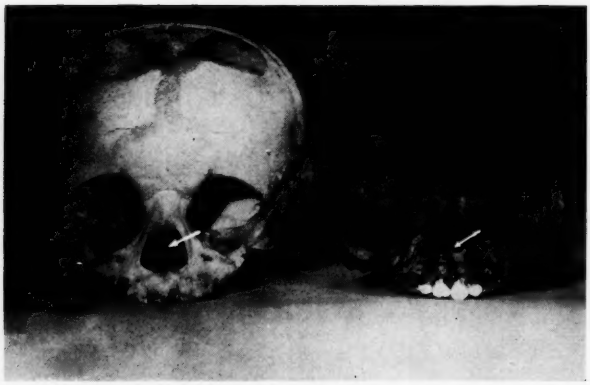

Fig. 2.—Shows the width of the vomerian groove at birth (1½ Millimeters) and the vulnerability of the septal cartilage to dislocation.

found that there was a variation from 1 to 2.5 cm. of outward projection of the nose.

In only one case was there an exception. In this case there was no variation between the occipito-frontal and the occipito-nasal measurement. The absence of outward projection of the nose in this case may be explained by the fact that this child happened to be a negro.

It appears that no one has given adequate consideration to the caudal portion of the nose in its relationship to any part of the skull nor the possibility of injury to that part during labor, whether it be normal or abnormal.

The most prevalent injury to the nose of the newly born is the dislocation of the cartilaginous portion of the septum from the vomerian groove (Fig. 2). In 23 infants which were examined, there were found two cases which showed dislocation of the septum from the vomerian groove and the columella. However, we are cognizant of the fact that many infants are born with noses flattened due to pressure during delivery, which eventually become straightened and elevated.

Autopsies were performed upon seven stillborn infants, employing the Kretchmann subperichondrial technique (an incision under the lip through the buccogingival fold). In inquiring into the

Fig. 3.—Shows method of tugging the tip of nose to reduce dislocated septal cartilage.

history it was learned that none of these deliveries had been instrumental and still anatomic displacement of the quadrangular cartilage was noted in two of the seven cases.

It is logical to assume that if there were two cases of displacement of the columella in 23 living infants, and that seven postmortems revealed two additional cases of septal dislocation, the incidence of nasal injury is fairly high either at birth or during labor.

Examination of 20 infants with a history of having been born through the cesarean route revealed that nine of these had the tests of labor, and that instruments had been applied before cesarean section. Upon examination these infants showed various degrees of septal deflections. Two had injuries during childhood and nine revealed straight septa. It may further be assumed that the nine children with the straight septa were the ones that did not go through the physiological tests of labor, while the other nine did. Therefore, injury to the nose may have had its inception at birth.

The obstetrician will always call for help and sometimes will take it upon himself to correct any external laceration or abrasion of the nose. Very few obstetricians look for intranasal deformities, such as cartilaginous dislocations of the septum from the vomerian ridge.

Fig. 4.—Visceral tenaculum employed for traction.

It is only when the infant has difficulty in nursing and there is some impairment in nasal respiration that the obstetrician or pediatrician becomes aware of an injury to the nose. Incidently, during the first week of birth the infant should normally breathe through his nose. However, if there has been an injury, he will learn with difficulty to breathe through his mouth.

It should be a routine practice for every obstetrician to inspect and palpate every infant's nose for septal deflections or nasal anatomical dislocations and fractures, just as routine examinations are conducted to determine the presence of any other deformities or injuries.

It is best to correct such deformity immediately before a greenstick fracture results or a callus forms. One has only to grasp the tip of the nose with the thumb and index finger, gently but firmly pulling it from side to side, or tugging the tip of the nose slightly ventrally (Fig. 3). One can usually feel or even hear the snap of the reduced dislocated cartilage.

In case this procedure or technique does not answer the purpose it might be suggested that a miniature Babcock visceral tenaculum or an Allis forceps be employed. These can be utilized for traction and manipulation to replace the dislocated cartilage.

Another instrument that may be employed for this purpose is the small Asch forceps consisting of two small blades on a hinge. The blades are placed into the nares on each side of the septum and by gently rocking the septum from side to side the cartilage will spring back into the vomerian groove.

Should the fracture or dislocation be neglected or overlooked and there be a lapse of several weeks, the obstetrician may be confronted with the additional problem of callus. The septum, when corrected, rests upon callus rather than in the vomerian groove, and it then becomes necessary to employ splints, tampons or packing in order to help keep the septum in its proper place.

Should the deformity be overlooked entirely, one may find in later life: a deflected septum with resultant impaired nasal respiration; trigger points with resultant nerve irritations, such as neuralgias or sphenopalatine ganglion irritations; faulty development of the upper jaw; sinusitis; the so-called adenoid facies which very often is due to anterior nasal obstruction, rather than choanal obstruction, and last but not least, the whole background becomes a panorama of "noseology" at adolescence. As the nose begins to grow, the external deformity becomes apparent by distortion, asymmetry, concavity or hump.

In conclusion, it is important to note that the incidence of septal deformity is widespread. Morrell MacKenzie discovered that there is asymmetry of nasal fossae in 77 per cent of 2000 skulls examined.

In our experience,[1] however, the percentage has been found to be lower. Of two hundred unselected adult patients who have consulted us as rhinologists because of various nasal complaints only nine required surgery of the septum.

Increased mental capacity of the race with greater brain development may account for a certain percentage of septal deflections and nasal deformity, but dystocia with all its elements may be a responsible factor for the high incidence of nasal injury and deflected septa. It is therefore strongly urged that such fractures be reduced promptly and that anatomic correction be made early, in order to reestablish proper nasal physiologic function.

Thanks are due to Drs. Kendall, Montgomery and Nelson for use of clinical material, and to Dr. Samuel Fomon for suggesting the subject.

1915 SPRUCE STREET.

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LI

PLASTIC SURGERY IN RECONSTRUCTING THE PARTIALLY ABSENT NOSE

AN ORIGINAL TECHNIQUE

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HOLLYWOOD, CALIF.

Skin grafts or pedicles should be used about the head or neck only when the defect is too large to be handled by other methods. Whenever possible, tissue absent in these areas is best replaced by adjusting adjacent tissue. Simple conservative methods should be considered primarily and the surgeon must keep in mind the expected end result. When a large scar is removed, the resulting raw area may be covered with the surrounding skin by utilizing a 'Z' flap or by employing adjacent relaxing incisions. Skin grafts may lose their pigment, wrinkle or contract.[2] Pedicles may be too thick and cosmetically unattractive. These facts must be considered when the surgical procedure is planned.

When the requirements of the case are such, a full thickness or thick split skin graft may be utilized.[1] Often a carefully planned pedicle is required.[3]

When the tip of the nose is absent because of injury or disease, it presents a specific problem. Restoration of the nasal lining, skin and often skeletal or cartilaginous structures must be good physiologically as well as cosmetically. The technique of repair is influenced by the size and type of deformity.[1] The plastic surgeon may choose a tube flap from the forehead, the neck or the arm. If the defect is small and involves the ala, an incision may be made above, a flap carefully undermined and rolled downward to form the new margin, and the resultant defect grafted.

From the Division of Reconstructive Plastic Surgery, Cedars of Lebanon Hospital, Hollywood, California.

Presented at a joint meeting of the Southwest Sections of the Los Angeles County Medical Association, November 1943, and by invitation at the U. S. Naval Hospital, Yosemite, California, January 2, 1944, in conjunction with colored surgical motion pictures.

Absence of tissue in the body of the nose may require other methods for repair. Again there must be restoration of both nasal lining and external skin. Careful planning of the operative procedure is mandatory. Whenever possible the adjacent tissue is utilized. Not only is the result more pleasing to the patient but so too is the economic burden.

REPORT OF CASES

The following cases, though possessing totally different problems, are presented because the identical procedure was utilized to gain tissue to reconstruct the nose.

In each case a rhinoplasty was carried out in conjunction with other measures. Thus a smaller nose allowed sufficient skin to repair the existing deformity.

CASE 1. B. R., white female, aged 30, was in an automobile accident two and one-half years prior to consultation. The face was cut by windshield glass, the latter severing portions of both the right and left nasal alae and causing long lacerations over the body of the nose, the glabella and the lip. (Fig. 1).

Examination. Examination revealed the scars to be somewhat keloidal in character (an operative scar and an old wound on other parts of the body were not keloidal). The injury on the left side had removed most of the lateral crus of the lower lateral cartilage, up to and including the arch. That on the right side had included the arch and a portion of the lateral crus of the lower lateral cartilage.

Surgery. Under local nerve block and infiltration anesthesia, incisions were made between the border of the upper and lower lateral cartilages, then brought forward and joined to separate the septum from the columella. The skin over the nose was undermined in a thick flap. The periosteum over the nasal bones was incised and deflected medially. The anterior aspect of the nasal arch was removed with right and left bayonet saws. The required amount of septum was removed both anteriorly and inferiorly. Through small stab incisions in the lateral aspect of the nasal vestibules, the periosteum over the nasal process of the maxillary bones was elevated; the latter were sawn through and the distal fragments reflected medially to form the new arch.

An incison was made over the glabella of the nose through the old scar and carried down toward the ala on the left side and only partially down on the right. With meticulous care the skin was

Fig. 1.—The patient sustained injury in an automobile accident two and one-half years ago with partial amputation of both ala nasi and numerous lacerations of the nose, lip and forehead. A rhinoplasty was done primarily to acquire tissue. A circular incision was made through an old scar on the nose, the flap meticulously dissected from the mucous membrane, then rolled downward to form the new alae. The resulting defect on the bridge was covered with a full thickness graft taken from behind the ear. At a second-stage operation a plate of rib necrocartilage was placed over the bridge and another piece inserted within the newly created left ala nasi.

separated from the mucous membrane and undermined down to the edge of the ala on both the right and left sides. This allowed the whole distal flap to be rolled downward to form the new tip. The columella was cut through the scar near the tip, a portion removed, then resewn to the tip and interdigitated as a "V". Through a separate incision along the lower border of the lower lateral cartilage on the right side, the latter was dissected free and a section removed without disturbing the nasal lining. A full thickness graft two inches long and three-fourths of an inch wide was taken from back of the left ear and grafted over the defect on the bridge of the nose.

Second Stage. Seven months later under local nerve block and infiltration anesthesia an incision was made between the upper and lower lateral cartilages on both sides, brought forward and joined, separating the septum from the columella. The skin over the nose including the previously placed full thickness graft, was thoroughly undermined. A plate of rib necrocartilage, two and one-fourth

Fig. 2.—The patient had cancer of the nose, apparently cured by cautery four years ago. Six months later patient consulted a "professor" who applied "cancer paste" which destroyed part of the nose, septum and nasal cartilages. A rhinoplasty was performed to gain tissue. A one-eighth inch circular flap around the cavity was turned inward to form the nasal lining, and nasolabial flaps were utilized to cover the external defect. A strut of rib necrocartilage was placed over the bridge to correct the saddle deformity. Another piece, sculptured to resemble the lateral crus of the lower lateral cartilage, was inserted within the right ala nasi.

inches long, seven-eighths of an inch wide and one-sixteenth of an inch thick was placed beneath the full thickness graft, the latter having adhered to the underlying tissues. A piece of rib necrocartilage sculptured to resemble the lateral crus of the lower lateral cartilage was placed within the border of the left ala through a small stab incision near the cheek. The graft extended from this point to the tip of the nose.

Three keloidal scars were removed, one on the lip, one on the glabella and one on the forehead.

CASE 2. A. R., white female, aged 69, had a small basal-cell carcinoma removed with an "electric needle" four years ago. Because the area healed slowly the patient consulted a "professor" who was "curing cancer" with paste. She applied the paste and within one month it caused part of the bone, cartilage and septum to "fall away." Within two weeks after the destruction the area healed cleanly and its character did not change up to the time of consultation. (Fig. 2).

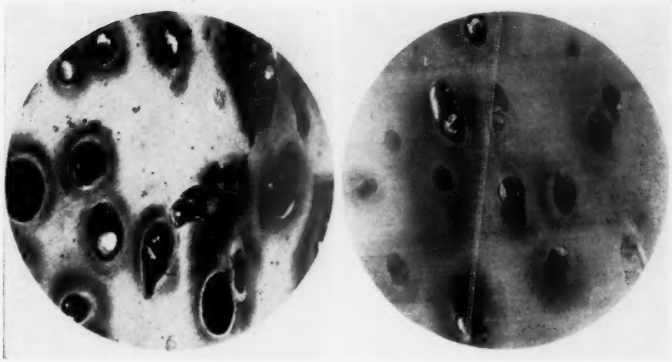

Fig. 3.—Left, Photomicrograph of rib necrocartilage which had been fixed in formalin solution for four days, then transferred to merthiolate-saline solution, 1:4. Two weeks later it was implanted in the anterior abdominal wall of the patient for seven days before removal for microscopic study. The cartilage is reasonably well preserved. Only at the periphery is there dissolution of the cartilage cells where the tissue stains pink. Centrally, the cells are large and take on a deep blue stain. The nuclei are prominent and in some places are vacuolated. x130.

Right, rib necrocartilage. The same piece of cartilage after having been left in the anterior abdominal wall for six months before removal for microscopic study. Though some of the cells show moderate vacuolation, the section as a whole presents well preserved cartilage cells. At the periphery, there is some dissolution of the cells and nuclei. In the center the nuclei are distinct and stain blue. x130.

Examination. Examination revealed a hole on the right side of the body of the nose with external dimensions slightly over one-half inch long and three-eighths inch wide. The area was clean and revealed the mucous membrane of the inside of the nose. Part of the nasal bone and the nasal process of the maxillary bone were destroyed, as was virtually all the right upper lateral cartilage. A section of the septum was absent causing a complete saddle. The upper wing of the lateral crus of the right lower lateral cartilage was missing.

Surgery. Under local anesthesia an incision was made between the upper and lower lateral cartilages on the left side, carried downward, joined with a somewhat similar incision on the right side to separate the columella from the septum, down to the nasal spine. The anterior aspect of the nasal arch was lowered slightly with a saw to allow for deflection of the bones. Through stab incisions in the lateral aspect of the vestibule on both sides the nasal process of the

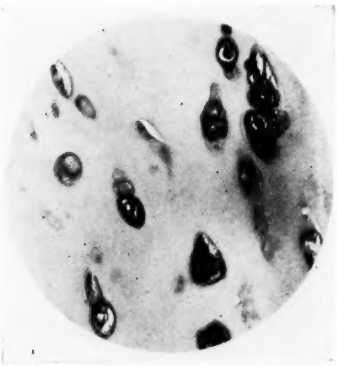

Fig. 4.—Photomicrograph of rib necrocartilage which has been fixed in formalin solution for four days, then transferred to merthiolate-saline solution, 1:4, and kept in the refrigerator for six months. The cartilage is as well preserved as that placed in the anterior abdominal wall. There is some loss of cytological detail at the periphery which takes on a pink stain, but the central portion stains blue and the nuclei and cells appear well preserved. x130.

maxillary bones were sawn through and the distal fragments reflected medially to form the new arch and narrow the nose. Through separate incisions below the borders of the lower lateral cartilages the latter were dissected free, and the required amount of cartilage was taken in the form of a hockey stick without disturbing the lining. The anterior aspect of the remaining septum was dissected to allow the tip of the nose closer approximation to the face.

A circular incision one-eighth of an inch distant from the wound crater was made, the flap undermined, turned inward and sutured across with triple O catgut, thus forming a lining. Incisions were then carried from the bridge of the nose downward some three inches on either side along the nasolabial fold, and these were joined by a single incision one inch long beginning just above the glabella and running downward to the center of the nose. These face flaps were thoroughly undermined over the maxillary area and then drawn over the body of the nose to cover the defect. The incisions were closed with interrupted "C" silk.

Second Stage. Eight and one-half months later the induration had subsided and under local anesthesia incisions were made between the upper and lower lateral cartilages, brought forward and joined

near the tip of the nose. The skin over the nose was undermined, including the previously reconstructed area. A strut of rib necrocartilage that had previously been prepared and modelled was inserted over the bridge of the nose to correct the saddle deformity. Another piece of rib necrocartilage was sculptured to simulate the lateral crus of the lower lateral cartilage. Through a small incision at the lateral aspect of the right ala a tunnel was created running to the tip of the nose to receive the graft. One-inch vaseline gauze packs were placed in either nostril and adhesive dressing support applied.

CARTILAGE

The cartilage used was taken from individuals in exitus and has been termed "rib necrocartilage".[4] It was treated by removing the perichondrium, placing it in formalin solution for four days, after which it was carefully washed and transferred to merthiolate-saline solution 1:4, then stored in the refrigerator. In order to follow the course of these implanted grafts, pieces of the same cartilage were transplanted to pockets immediately beneath the skin of the anterior abdominal wall. These latter were removed chronologically up to six months and studied microscopically. The sections showed well preserved cartilage cells, in some areas the cells were vacuolated, but mainly they were well preserved with distinct nuclei. There was also invasion of some fibrotic elements especially in the periphery. (Figs. 3 and 4). Grossly the specimens were virtually the same size as that originally implanted.

Since the completion of this paper rib necrocartilage implanted in the anterior abdominal wall for fifteen months has been removed and presents virtually the same microscopic picture.

DISCUSSION

Both cases healed uneventfully. By performing a rhinoplasty and thus making the skeletal and cartilaginous structures of the nose smaller, tissue was gained. This in conjunction with borrowing skin from adjacent areas allowed for reparative procedures that might not have been possible otherwise.

By utilizing necrocartilage, defects were filled and the contour of the nose was made more exact. Though the cartilage may be invaded by fibrous elements, these latter act as struts appreciably as strong as the cartilage itself. Therefore, necrocartilage in selected cases allows for an excellent method of replacing lost tissues.

CONCLUSIONS

1. A new procedure is presented for reconstructing the partially absent nose by utilizing rhinoplastic methods.

2. The course of necrocartilage in the human has been followed microscopically and its use in plastic procedures presented.

6777 HOLLYWOOD BOULEVARD.

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Clinical Notes

LII

CASE REPORT OF A CAROTID BODY TUMOR

PIERRE VIOLÉ, M.D.

LOS ANGELES, CALIF.

Experience with this tumor has been chiefly confined to the past fifty years, during which time a total of only some 250 cases have been reported. These were largely individual cases; hence the experience of any one operator has been limited, and literature pertaining to it is scant.

Marchand in 1891 appears to have been the first to fully describe this neoplasm, after excision of a carotid body tumor by Riegner in 1880. Through the succeeding decade a scattered few cases made their way into medical literature, and in 1903 Scudder[1] reported a case diagnosed as "perithelioma", a tumor of nine years' standing, which was successfully removed. This was the first case of surgery of a carotid body tumor authentically reported in this country.

Relatively little was known of the function of this gland until about 1930; it had not been considered essential to well-being or even to life itself. In more recent years it has been demonstrated that the respiration and circulation are influenced by the carotid body, due to chemoreceptors present in the gland which react to arterial blood changes. Schmidt and Comroe[2] state more conclusively that the role played by carotid bodies, is a significant one in the control of respiration, and that changes in chemical composition of the blood affect the respiration through both the carotid body and the respiratory center itself. Twombly, Simon and Steinberger,[3] in an article on this subject published in 1943, state that experiments show the carotid

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From the Department of Otolaryngology of the University of Southern California School of Medicine.

Read before the Sixty-Sixth Annual Meeting of the American Laryngological Association, New York, N. Y., June 7-8, 1944.

body to be more sensitive than the respiratory center to the respiratory poisons, such as nicotine, cyanide and lobeline and to anoxia, whereas the center itself is more sensitive to changes in carbon dioxide content and pH of the blood.

As is true of other tumors, their cause is not known. It is thought there may be a hereditary factor in the development of this growth, since a number of familial cases have been reported. Both sexes seem to be equally susceptible, both white and negroid races are involved, the age of patients varying from early childhood to senility.

These tumors develop slowly, usually over a period of years. Often the only symptom noticed by the patient is an external swelling in the area of the gland itself, which may appear to remain almost static over a prolonged time, until a sudden acceleration may suggest medical investigation. The growth is usually round or oval, smooth, and rather elastic though compressible. It is movable from side to side but not up and down. The tumor may not be apparent at first glance, but with the head extended, close inspection under a good illuminator may reveal a swelling behind and below the angle of the jaw. Eventually pressure due to the growth may produce some hoarseness, cough, possible nausea, vomiting or tinnitus, and occasionally local pain is experienced. However, these patients are usually symptom-free for many years before such developments call the tumor to their serious attention.

This neoplasm is predominantly unilateral, though a few cases have been reported where the patient had a growth on both sides of the neck. These may occur simultaneously, or one may develop after removal of the other, sometimes a number of years subsequent to the original operation. In the discussion of this subject by Gratiot[4] he cites a case reported by Lund in which the right gland was removed 29 years following excision of the left. He adds that 15 to 20 per cent of carotid body tumors become locally malignant after remaining benign for years, with only rare metastases in the regional lymph nodes, distant metastases being unconfirmed.

Without the somewhat precarious procedure of aspiration biopsy, pre-operative carotid body tumor is difficult to diagnose. It is often logically confused with other cervical conditions, such as inflammatory lymph nodes, tubercular nodes, malignant lymphomas, branchial-cleft cysts, aberrant thyroid tissue, lipoma and sarcoma. Gratiot differentiates it from such other growths by the fact that it is the only "single, solid, slow-growing tumor, intimately attached to the

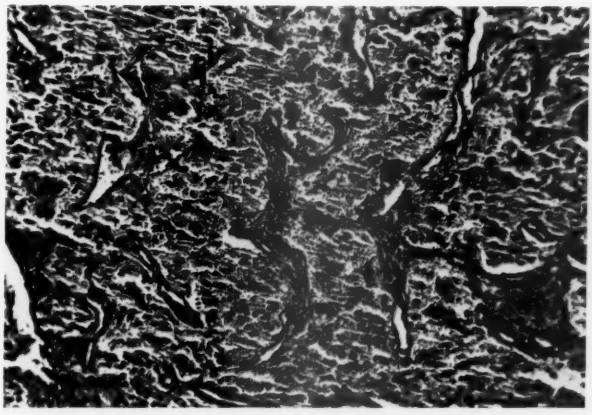

Fig. 1.—Photomicrograph of carotid body tumor revealing characteristic paraganglioma formation.

carotid bifurcation," and concludes that exploration may be the only recourse left to the operator to establish an accurate diagnosis. Laboratory findings offer little conclusive evidence, as blood, urine and x-ray examinations may all be negative in the presence of the tumor. Surgical procedure is the universally accepted treatment. In the article above referred to by Twombly et al., they state that perhaps the most important point in diagnosis is to consider any slow-growing cervical mass as a possible carotid body tumor.

Ewing[5] considers this neoplasm in a class by itself histologically, describing the structure as complex and in general that of an alveolar perithelioma, but varying in many details. He states a skillful enucleation is usually successful in preventing recurrence, but cites an estimated 35 per cent mortality from the operation.

J. C., a Japanese male, 59 years of age, entered Los Angeles General Hospital September 3, 1942, complaining of a swelling in the right side of his neck of three months' duration. Apparently he had been in good health until the onset of this condition, when he became conscious of difficulty in swallowing and diminished appetite.

Just prior to his appearance at the hospital he had been seen in New Mexico, at which time a biopsy was made and a tentative diagnosis of carcinoma was recorded.

The past history was noncontributory.

Physical examination revealed a Japanese adult in no apparent distress, the only significant findings being as follows:

A 4 x 6 cm. mass projected into the naso- and oro-pharynx from the right lateral pharyngeal wall. The mucosa over the mass appeared normal. The mass occupied about one-third of the oro- and naso-pharynx, and seemed soft but nonfluctuant on palpation.

In the neck at the upper portion of the anterior border of the right sternocleidomastoid muscle a firm mass was palpable, which moved with swallowing.

No other glands were palpable. The urine and the blood were negative.

Three biopsies were attempted at the hospital. We were unable to corroborate the biopsy previously made and were not able to make any diagnosis.

The results of the biopsies were:

Sept. 17, 1942—Miscellaneous pharyngeal mucous membrane, glands and muscle. No pathological diagnosis.

Sept. 26, 1942—Biopsy attempted by dissection of neck. No tissue obtained for section.

Oct. 5, 1942—Pharynx section showed only connective tissue, many blood vessels, and some adipose tissue. Tumor could not be diagnosed.

On October 14, 1942, the patient was taken to the operating room for excision of the pharyngeal tumor. The operative procedure consisted of ligation of the right common carotid to control hemorrhage, and as the tumor presented itself primarily in the pharynx, it was decided to enucleate the mass through the oral approach. The mass was semi-soft, moderately fluctuant, and movable from side to side. The enucleation was accompanied by considerable bleeding.

The patient was returned to bed in a state of shock and, in spite of all supportive measures attempted, expired within 24 hours following surgery.

The specimen consisted of an irregularly oval portion of tissue 4 x 3.5 x 1.8 cm. The tissue was yellow-brown in color and presented numerous fibrous bands on all aspects. On section a mottled yellow-brown appearance was observed, suggesting a rather fatty component, although its firm consistency argued against this possibility.

Microscopic examination revealed a tumor having the typical structure of a "paraganglioma" of the carotid body. There was no

evidence of malignancy. A very thorough autopsy was performed. No metastatic lesions were encountered which could be related to the tumor in question.

SUMMARY

This case of a carotid body tumor, also diagnosed as "paraganglioma of the carotid body", is presented primarily because of the relatively infrequent occurrence of this type of tumor, the problem of differential diagnosis which it presents, and the seriousness of operative procedure.

1930 Wilshire Boulevard.

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LIII

CONGENITAL LYMPHANGIOMATOUS MACROGLOSSIA WITH CYSTIC HYGROMA OF THE NECK

D. M. Lierle, M.D.

Iowa City, Iowa

This presentation consists of a brief discussion of congenital lymphangiomatous macroglossia, with cystic hygroma of the neck. It includes a report of one case which has been treated, and two more which are receiving treatment at the present time.

Cystic hygroma may be congenital or acquired. Its origin frequently is in the submaxillary region, and it may involve the face, the floor of the mouth, and the tongue. The essential pathology consists of a dilatation of the lymphatic spaces. This dilatation is due to obstruction caused by the absence of efferent lymph vessels, or by lymphatics which do not have patent channels. Sich believes that the origin of lymphangiomas is in an isolated vessel or a connective tissue primitive rest which perforates into new lymph vessels. The hygroma usually consists of multilocular cysts lined with endothelium, with fibromuscular walls. These cysts are usually translucent and contain lymph fluid, but they also may have milky or chocolate-colored fluid with cholesterin.

Sections of the tongue in lymphangiomatous macroglossia reveal excess fibrous tissue, lymph vessels surrounded by enlarged vessels containing coagulated blood and lymphadenomatous new growth. Fibrous tissue, according to Buttin,[1] may cause muscle fibers to degenerate by pushing them aside and exerting pressure upon them. Consequently only fibrous tissue and large lymph spaces containing blood and lymph may be seen.

The cases in this series were three girls, eleven months, two and three years old, respectively, when admitted to the hospital. All had varying degrees of macroglossia at birth. Two had associated cystic hygroma of the neck at birth. According to the history of the third, involvement of the neck did not occur until several months

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after birth. Just previous to hospitalization, the macroglossia and the cysts of the neck began to increase rapidly in size in each case.

Case 1 was first admitted to the hospital at the age of 11 months. The tongue was so large that it could not be retained in the mouth. The obstruction was so great that there was marked difficulty in eating and breathing. The mouth was constantly open, and there was dribbling of saliva. The lower jaw was deformed and the lower lip slightly hypertrophied. The tongue, exposed to the air, had become hard and fissured, and there was hypertrophy of the epithelium. Several areas of bluish discoloration could be seen and there was superficial ulceration of the mucous membrane, causing considerable discomfort.

The treatment consisted of surgical removal of the tumor of the neck. This appeared to arise from the left submaxillary region. It involved the floor of the mouth and extended into the base of the tongue. Since x-ray treatment in angiomata causes swelling of the endothelial cells and a degeneration of the elastic fibers and muscle with a replacement by fibrous tissue, the tongue was treated with divided doses of x-ray. A total of about 3000 roentgens, divided into doses of 400 to 500 roentgens, was used over a period of about four months. For only a short time, there was a definite decrease in the size of the tongue. Later it increased somewhat in size.

Later, a plastic operation was done on the tongue. The anterior portion was amputated and a large wedge-shaped section was removed, so that the tongue was practically normal in size. Microscopic sections revealed the absence of lymph spaces, but there was degeneration of muscle with replacement by a large amount of fibrous tissue. There has been no recurrence in a period of four years.

Cases 2 and 3 are in the hospital at the present time receiving similar treatment.

UNIVERSITY HOSPITAL.

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LIV

HYDROSTATIC PRESSURE IN ADENOID HEMORRHAGE

HEMAN GRANT, M.D.

DETROIT, MICH.

Thousands of operations are performed yearly upon the adenoid tissue. It is done indiscriminately by most of our surgeons. We rather expect an occasional hemorrhage from the tonsil bed and are prepared to meet it, but we give little thought to the adenoid bleeding because it always seems so easily controlled. How many of us would know what to do if confronted with an impending fatal hemorrhage from an aberrant artery coming through the basilar process of the occipital bone? In a conference with one of our most capable and distinguished men he mentioned three such experiences in which he did not know what to do next. One of our authors speaks about his personal dilemma in a case which required anterior and postnasal plugging for six days. Any procedure which will simplify this problem should be made common knowledge.

Regional Anatomy. The pharyngeal tonsil lies anterior to the basilar process of the occipital bone. This process joins anteriorly with the body of the sphenoid. Until the age of puberty there exists a cartilaginous union traversed by vessels. Laterally the petro-occipital suture separates the basilar process from the petrous portion of the temporal bone and this is traversed by larger vessels. Our textbooks and anatomies make no mention of the vascular supply to the adenoid structure. However, in a study of the arterial supply itself one is struck by the numerous vessels distributed to this region by the external and internal carotids and the vertebral artery, their anastomoses and variations. Piersol[1] says:

> "In its cervical portion the internal carotid occasionally takes a somewhat sinuous course, and, especially in its upper part, may be thrown into a pronounced horseshoe curve. It may give rise to branches which normally spring from the external carotid, as, for example, the ascending pharyngeal and lingual, and *accessory branches may arise from its intracranial portion.*"

Some of our problems may come from this latter source by way of accessory branches and emissary vessels which penetrate the bone and sutures.

A normal boy, S. W., aged 6, entered Grace Hospital May 22, 1942. Removal of tonsils and adenoids was uneventful and he left the operating table with what was thought to be the usual oozing from the nasopharynx. That evening nasal packing seemed indicated.

The following morning there was marked pallor and signs of continued bleeding. The throat was inspected and found normal. Bleeding seemed to come chiefly from the nose. Anterior and postnasal plugging was resorted to with only temporary success. The hemoglobin dropped below 50 per cent and the outlook became serious. Blood transfusion was ordered and the patient returned to the operating room where upon close inspection the source of bleeding was located in a sulcus at the center of the adenoid site. This seemed to lead directly into the basilar process. Firm pressure controlled it but after one-half hour the bleeding recurred when pressure was released. Then hydrostatic pressure was used. With a long needle about 20 cc. of physiological sodium chloride solution was forced into the mucous membranes above and at the side which ballooned the entire area together. All bleeding stopped instantly and there was no recurrence. We were a little alarmed at what this ballooning process might do if it extended into the larynx and a word of caution should be used in this connection.

If this procedure turns out to be as successful in similar cases it will be a most worthwhile addition to our control of nose and throat hemorrhages. It is the first time that such a method has been used in this connection to my knowledge and to that of the numerous surgeons interviewed. Full credit for the suggestion should go to Dr. Himelhoch, the family physician, who had used the method in a throat hemorrhage.

10 PETERBORO.

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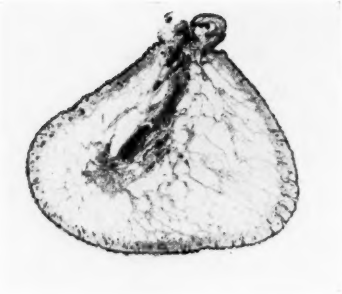

Section of Cyst (x3).

LV

CONGENITAL DERMOID TUMOR OF THE NASOPHARYNX

JOHN H. FOSTER, M.D.

HOUSTON, TEXAS

J. E. H., aged 19 days, was referred to me February 25, 1944. The mother said that there had been some obstruction in breathing since birth and that in a short time she noticed that the infant got something in its mouth and suckled on it. The baby was taken to a physician who discovered a tumor in its throat and referred them to me.

Examination showed a well-nourished baby apparently well. On depressing the tongue a rounded tumor was visible attached to the posterior pharyngeal wall. It was freely movable on a small pedicle and at times disappeared into the nasopharynx; at others came into the mouth.

On account of the pedicle's being so slender it was deemed safe to remove the tumor. It was grasped with forceps and removed with a snare. There was no bleeding of any consequence.

The tumor was sent to Dr. Violet Keiller who reported as follows:

Read before the Sixty-Sixth Annual Meeting of the American Laryngological Association, New York, N. Y., June 8, 1944.

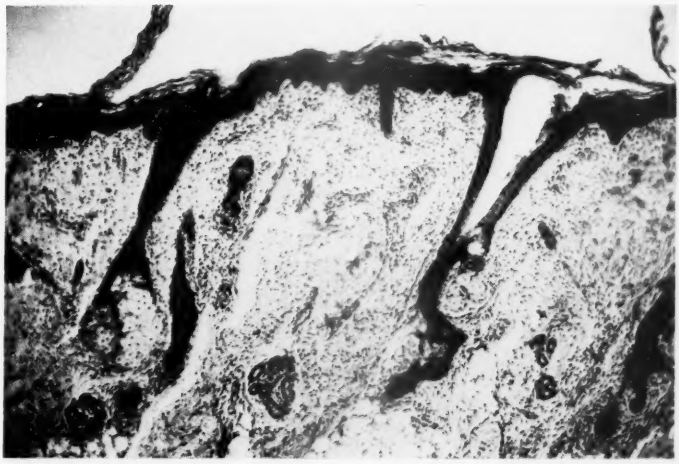

Fig. 2.—High Power Section.

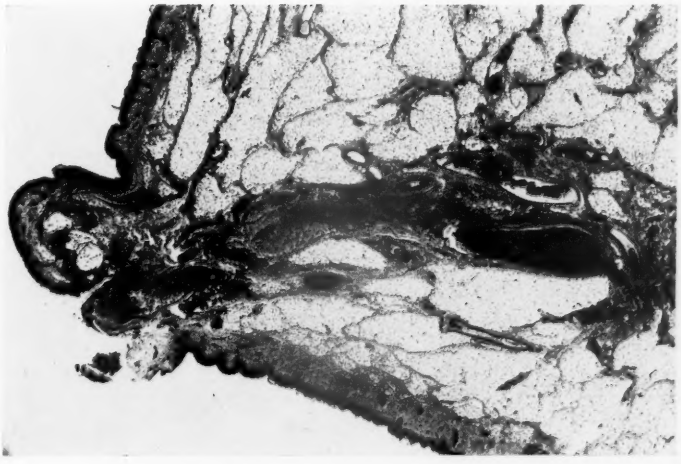

Fig. 3.—Section Through the Pedicle.

"*Gross*: Tumor removed from the posterior pharyngeal wall, about the level of the hard palate. It was connected with the pharyngeal wall by a narrow pedicle, of rather soft tissue, 4 mm. in diameter and 2.2 mm. long (as removed). The tumor is bean-shaped—a rather fat bean, 1.5 cm. long by 1.2 cm. by 1 cm., and the pedicle is placed like the stalk of a bean but closer to one end than to the other. The pedicle seems to have a small hollow in its center. The surface of the tumor (but not the stalk) is delicate integument, with tiny pits visible with a hand lens and minute hairs just perceptible to the naked eye. The specimen floats in water, and that and its texture suggest that most or perhaps all of the interior is composed of fat tissue.

"*Microscopic*: Epidermis with a thick keratin layer, partly macerated, covers the tumor, except that at a small area near the pedicle there is, on one side of this, a covering of columnar "nasal" epithelium. Here there are a few mucous glands. In the tumor proper, sebaceous and sweat glands are present in orderly arrangement, and minute hair follicles are seen. Fat forms the greater part of the interior of the mass, and in the very center there is a slender bar of cartilage, pointing to the base of the stalk but not entering this. The stalk consists of fibrous tissue, with one or two bundles of nerve fibers—no ganglion cells—and with large, thin walled vessels. There is no evidence of active growth.

Diagnosis: Congenital nasopharyngeal dermoid."

COMMENT

This case is reported simply as an anomaly found in so young an infant.

1304 WALKER AVENUE.

LVI

CONGENITAL ANTEFLEXION OF THE UVULA ACCOMPANIED BY A SWELLING OF THE SOFT PALATE; ITS CORRECTION BY PLASTIC METHODS

ERNEST WODAK, M.D.

TEL-AVIV, PALESTINE

A 24-year-old female patient (J. G.) consulted me regarding a serious impediment of speech which her parents stated had existed from her earliest childhood without known cause. Previous examinations had not yielded any constructive advice for the relief of this impediment.

Examination. The hard palate was normal. The soft palate was somewhat thickened, with movements normal, though somewhat restricted. The uvula could not be identified as such, but at its base there was a bulge about the size and thickness of a button. My impression was that the thickening about this area was caused by the anteflexion and adhesion of the uvula to the soft palate.

Function. There was a very striking rhinolalia aperta present, the patient's speech being scarcely intelligible. She was unable to put out a burning candle by blowing. My recommendation was to free the adherent uvula and to restore it to its normal position.

Procedure. A traverse incision was made along the upper edge of the button-like thickening, thus freeing the tip of the uvula from the area of the base and pulling it downward to its normal position. After undermining both edges, the rhombus-shaped incision was closed by horizontal sutures. I thus prevented the adhesion of the scarred uvula to its original position. Care was taken not to perforate the soft palate.

Postoperative Course. This was uneventful except for a marked swelling of the uvula. The sutures were removed after five days, at which time the uvula was normal in size and in its proper position. After two weeks the patient and her parents stated that her speech was considerably improved, especially for gutteral sounds which were formerly completely unintelligible.

While the patient is satisfied with the results so far, I have recommended a course of exercises under the guidance of an experienced speech teacher. If this does not accomplish the desired result, a pharyngo-plastic retro-displacement of the entire soft palate must be considered.

I am reporting this case, because during 24 years of plastic work, I have not seen one similar to it, nor have I been able to find a report of one in the biblography now at my disposal.

86A ROTSHILD BOULEVARD.

LVII

A CASE OF MALIGNANT RECURRING NASAL HEMORRHAGE OF UNDETERMINED ETIOLOGY

SAMUEL SALINGER, M.D.

CHICAGO, ILL.

I am reporting a case of daily recurring epistaxis in a woman of 23, which has been under my observation for the past three years. Despite every possible pathologic and hematologic test to which I and my colleagues have subjected her, we have been unable to arrive at a satisfactory diagnosis.

I first saw the young lady in July, 1941, when she gave a history of nosebleeds alternating between the two sides over a period of years, increasing in frequency until they were a daily occurrence. Examination at that time revealed a hyperemia of both Kiesselbach areas. Despite repeated cauterizations, the bleeding continued. Within a month or two an ulcer developed on the right side of the septum which bled repeatedly despite frequent cauterizations and soon eroded through the septum. The nasal chambers were otherwise normal as was the blood examination.

In December, 1941, a submucous resection was performed, which was attended by average bleeding. Since the hemorrhages continued, she was referred to an internist who reported only a moderate degree of secondary anemia. X-ray examination failed to reveal any abnormality. Radon paste was packed into the nose several times without result. It was noted at this time that the septum on the right side posterior to the perforation was spongy and swollen. A specimen taken for biopsy from this region was reported as "fibrin with many polymorphonuclear leucocytes; no tumor cells seen." She was given large doses of cevitamic acid and vitamin K without result. Since the hemorrhages persisted, it was decided to try radium. She received a number of radium treatments within the nose totalling 600 mg. hrs.

Read before the Sixty-Sixth Annual Meeting of the American Laryngological Association, New York, N. Y., June 8, 1944.

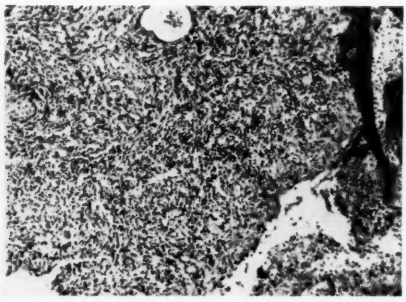

Fig. 1.—Section from septal ulceration. Granulation tissue with foci of necrosis; no evidence of malignancy.

X-ray films and planagraphs taken in April, 1942, disclosed an abnormality in the right maxillary sinus. A Caldwell-Luc operation was performed, revealing a very small antrum confined mostly to the nasal side and containing a mass of spongy tissue which was thoroughly cleaned out. Histologic examination disclosed "chronic inflammation with granulation tissue." The sinus healed promptly, but the hemorrhages persisted and the granulomatous area in the right side of the septum was still present. A specimen taken for biopsy was reported as "granulation tissue with foci of necrosis." She was given an additional 300 mg. hrs. of radium intranasally. The bacteriologic reports were negative for non-aerobic bacteria. Guinea pig inoculation for tuberculosis was also negative. We assumed then that the hemorrhage was due to self trauma, and referred her to a neuropsychiatrist who was unable to confirm our suspicion.

It is interesting to note that despite repeated examinations we were never able to pick out a definite bleeding point, nor was it possible to induce hemorrhage except by direct trauma with a metal instrument.

Another interesting point is the fact that the patient frequently complained of a throbbing sensation in the bridge of the nose, and at times exhibited a cyanotic swollen area which was tender to the touch and which at times would disappear completely. Radium was again applied, and an atrophy of all the nasal structures was beginning to be manifest.

The patient was hospitalized several times in 1942 and 1943, and was examined and treated by our hematologist, Dr. Raphael

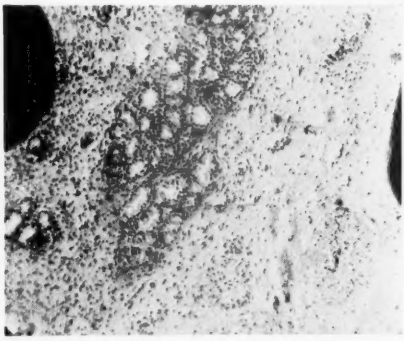

Fig. 2.—Tissue from maxillary sinus. Chronic inflammation with granulation tissue.

Isaacs, who reported nothing abnormal in the blood except a marked secondary anemia.

On March 6, 1943, we removed a wedge of skin, bone and mucous membrane constituting the swollen nasal prominence, and submitted the tissue for histologic and bacteriologic study. The report was "organization tissue with periosteal and endosteal fibrosis and new formation of bone." It was negative for tuberculosis sarcoid, or carcinoma. The cultures were reported as staphylococcus, hemolytic streptococcus and anhemolytic streptococcus and staphylococcus.

While in the hospital a search was made among her effects, and on two occasions a safety pin was found which on being tested with benzedrine gave a positive reaction for blood. The patient denied self trauma, and repeated examinations by our neuropsychiatrist, Dr. Gitelson, failed to elicit the information we desired. It was noted now that the evidences of trauma were repeatedly seen on the left inferior turbinate and the posterior margin of the large septal perforation, the only two areas readily accessible to a right-handed person. The wound over the nasal bridge healed and reopened frequently. When sealed with a metal cap it would heal completely, but would reopen when exposed again. Examination of this lesion by our dermatologist was reported as simple ulcer of probable traumatic origin.

At times the nose was packed with gauze saturated in zinc peroxide and the entire nose covered with a metal cap for two to three days at a time. During such periods there was no bleeding.

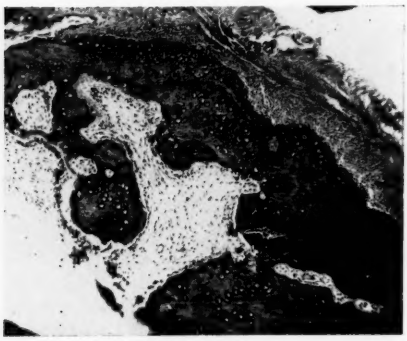

Fig. 3.—Tissue from nasal dorsum. Organization tissue with periosteal and endosteal fibrosis and new formation of bone.

Despite a number of transfusions the blood count has remained low (hemoglobin around 50% and red blood count, 3,000,000). The patient has developed an allergy to blood and now has to depend on hematinics and liver.

We are convinced that the bleeding is the result of self trauma, but are unable to obtain a confession from the patient. She is definitely not a schizophrenic and thus far can be classed merely as a hysteric with strong fixations. We feel certain that we have eliminated tuberculous lues, malignant granuloma, sarcoid, Osler's disease, carcinoma, sarcoma, anaerobic infection, lupus, mycotic infection and all blood dyscrasias, and can only conclude that the case is one of self-inflicted trauma based on as yet undisclosed psychogenic impulses.

The report of the psychiatrist, Dr. Maxwell Gitelson, is as follows:

"This patient has been studied for nearly two years, beginning with ordinary interview techniques. Blood pressure readings were taken during a number of the interviews and revealed an average increase in the systolic pressure from 120 to 140 at certain critical points in the interviews. But no bleeding occurred at these points. There were two interviews conducted with the patient attached to a Keeler polygraph. Significant increases in the pulse pressures were noted during these interviews at pertinent points in the interrogation. But no bleeding occurred at these times. A sodium

amytal interview was finally conducted and at this time it was made clearly evident that the patient bore great hostility to her father who was a drunkard and whose practice it had been in her childhood to tweak her nose during moments of erotized friendliness towards her. She recalled great fear of her father when during his periods of intoxication he would pursue her and her mother with a knife.

"Because of this evidence of specifically connected hostility and anxiety and because of the failure to elicit this hostility in face-to-face interviews I have been attempting to carry on a psychotherapy of the patient under hypnosis since January 18, 1944. This was undertaken in the hope that she might be better able to ventilate her feelings with the critical functions of the ego suspended. She has now been hypnotized over a period of six weeks.

"As is obvious from the clinical facts, there has been no effect of this treatment on the patient's major complaint. Interesting discoveries have been made; for example, the patient adheres as rigidly to her defensive symptoms under hypnosis as do patients normally without benefit of hypnosis. This is contrary to the general impression that hypnosis is supposed to reduce the resistance of the individual. Certainly, this girl is the most resistive individual I have ever encountered despite the fact that she goes into a profound trance, the depth of which is verified by the fact of complete post-hypnotic amnesia.

"The work has been largely directed towards finding out what the directions and intensities of her hostilities are which would make it necessary for her to turn so much self-destructiveness on herself. It seems that there is a great deal of envy of her father, whose drunkenness and bad behavior, failure to support the family, were too easily forgiven by the mother. Nevertheless, despite all these actual reasons in the life situation, the patient's rigid moral and ethical attitudes will not permit her to allow any experience or recognition in herself of the fact of resentment, even less giving vent to it in any degree. However, this is the direction in which treatment to this point has gone.

"I am entertaining no illusions as to the prognosis psychiatrically in this case which remains extremely guarded, especially in view of the fact that a diagnosis of the presenting symptoms as such has not been made, and in view of the fact that the mechanism of the induction of bleeding on the basis of psychic tensions is in no way clear."

At the present time (June 21, 1944) the patient is again in the hospital. Her red count is under 2,000,000, hemoglobin, 30 per cent. There are no new findings. The patient is receiving injections of packed cells and will be subjected to further psychoanalysis.

25 East Washington Street.

LVIII

THE USE OF A PERMANENT (ALNICO) MAGNET IN THE PERORAL REMOVAL OF A METALLIC FOREIGN BODY (PADLOCK) FROM THE STOMACH

SAMUEL SILBER, M.D.,

CARL KAPLAN, M.D.

AND

BERNARD EPSTEIN, M.D.

BROOKLYN, N. Y.

The first definitely planned stomach operation, according to Finney,[1] was the case reported by Crollius in 1602. The extraction by Mathis then, of a knife which had slipped into the stomach of a peasant who practiced jugglery, also marked the first surgical removal of a foreign body from the stomach. Of interest also, from our viewpoint, was the suggested use of a magnet over the abdomen in the stomach region. Until comparatively recently gastrotomy has remained the accepted procedure for the removal of swallowed foreign bodies.

We believe the case reported here is the first recorded in which a permanent magnet was used for the peroral extraction of a metallic foreign body from the stomach. Grob[3] recently used a small cylindrical solenoid for similar purposes. The method, however, is more cumbersome and not as readily or widely applicable as ours. The object removed in our case was a small, bulky padlock, and the ease with which it was removed immediately suggests the possibility of the application of the technic to the removal of other frequently encountered foreign bodies.

REPORT OF A CASE

D. B., a 5½-year-old boy was seen by us eight days after he had swallowed a small padlock. Fluoroscopic and radiographic ex-

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Case presented and discussed by the authors at a meeting of the Section on Laryngology, Rhinology and Otology of the Medical Society of the County of Kings and Academy of Medicine of Brooklyn, Nov. 10, 1943.

Fig. 1

amination immediately after the accident revealed the lock in the stomach.

After a week's trial of antispasmodics and bulk meals, the child was referred to a surgeon for gastrotomy. Before resorting to surgery, however, the parents brought the youngster to one of us for pediatric consultation. Fluoroscopy showed the padlock to be freely movable in the stomach. The possibility that a magnet might be employed for the peroral removal of the lock suggested itself, and the parents were instructed to provide an exact duplicate of the ingested lock. The lock measured 1 x 1¼ x ¼ inches, the bow measured an inch, and it was easily attracted by a permanent bar magnet.

The General Electric Company generously provided a new type of permanent magnet designated as "Alnico," which has the capacity of lifting 60 times its own weight. It is composed of an alloy of aluminum, nickel, cobalt and iron. The magnet used was one

inch long and one-eighth inch in diameter. This was grooved at one end, firmly attached to a Levine tube and a firm cord was passed around the tube over the groove to reenforce the attachment (Fig. 1).

The magnet was passed into the stomach under fluoroscopic control, and contact was made with the bow of the magnet after a few attempts. When the lower side was attracted the attraction was easily broken, when desired, by sharply pulling the tube so that the lock came in sudden contact with the cardiac sphincter.

After the desired contact had been achieved the table was tilted so that the patient, resting on his right side, was lying at a 60° angle with his head downward, thus helping to overcome the resistance of the "esophageal pinchcock" at the cardiac orifice. The tube was then carefully pulled so that the lock passed into the esophagus by maintenance of the steady pull on the magnet. When the lock was in the distal esophagus an attempt was made to pull it further proximally, with the intention of extracting it through the patient's mouth. Successful elevation of the lock beyond the region of the esophageal constriction at the bifurcation of the trachea was effected. At the level of the cricoid cartilage, however, the cricopharyngeal sphincter was sufficiently strong to balk further progress of the lock despite repeated attempts. Mosher mentions that the crico-pharyngeal sphincter is strong enough to pull a foreign body such as a coin from the grasp of a forceps if it is not firmly held.[1]

At this point the endoscopist was called upon to remove the lock through an esophagoscope. However, the child began to vomit and the procedure had to be halted. The lock was pushed back into the stomach and the attempt was abandoned for the day.

The next day, after proper premedication, the magnet was again easily introduced into the stomach, and the lock drawn past the cardio-esophageal orifice into the proximal third of the esophagus. Spot roentgenograms were taken of the procedure showing the position of the magnet and the lock before contact (Fig. 2), after contact (Fig. 3), and with the lock in the esophagus (Fig. 4). When the lock was in the esophagus the esophagoscope was passed and the lock withdrawn by means of a Jackson dangling forceps. The entire procedure took about four minutes. The child was returned to his room and two hours later was enjoying his supper.

The padlock was corroded by the gastric juices and none of the original lacquer remained.

Fig. 2

Fig. 3

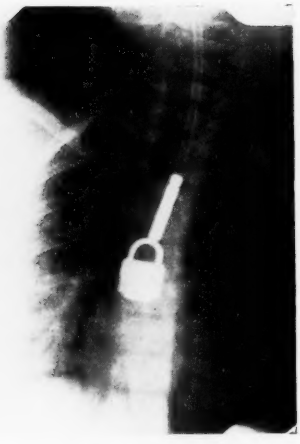

Fig. 4

DISCUSSION

The most important question to answer after the ingestion of a foreign body, is whether or not it can pass through the intestinal canal without difficulty or danger. There are numerous instances in which coins, safety pins, nails, small toys and similar objects have been evacuated normally.[5, 12] Occasionally large and unusual foreign bodies have been passed.[7, 8] Nevertheless, reports have been made in which perforation,[9-12] obstruction, and pressure necrosis[12] have occurred. Usually the advice of Jackson is accepted. He recommends waiting about a week for normal passage and then taking steps to remove the foreign body endoscopically if possible.[6] Of course, perforation or obstruction is a surgical problem.

With the acceptance of peroral endoscopy as the most effective approach to the removal of foreign bodies from the food or air passages, individualized procedures have been developed for the solution of various problems, and numerous types of ingenious grasping forceps have been originated. The use of the magnet may simplify many of these procedures but not all metallic objects are attracted by a magnet. The technic described here would apply only to objects made of iron, steel or nickel. When required, the coercive power of the Alnico magnet could be enhanced by concentrating the magnetic flux through a steel sheath about the magnet.

The use of a fixed magnet can be extended to many cases of a similar nature. Alnico magnets could be passed through a rubber tube of sufficient diameter into the food or air passages, and less bulky attractable objects such as hairpins, straight and safety pins, nails, or tacks, could be drawn into the lumen of the tube. Then the tube could be withdrawn perorally, thus obviating the possibility of trauma. All this would have to be done under fluoroscopic control. The Alnico magnet could also be used as one blade of a grasping forceps, the other blade being made of a nonmagnetic metal. This forceps could be used in the air as well as food passages, and the safety of the procedure would be enhanced by attracting the foreign body away from the walls of the confining passage.

The possibilities of the method described are being further explored at present.

SUMMARY

1. The peroral removal of a metallic foreign body from the stomach by means of a permanent (Alnico) magnet is described.

2. The parts played by fluoroscopy and esophageal endoscopy in this procedure are noted.

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Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, March 6, 1944

THE PRESIDENT, DR. SAMUEL J. PEARLMAN, IN THE CHAIR

(The Scientific Program was presented by the Department of Otolaryngology of the University of Illinois)

Influence of Aging on the Nasal Mucosa: An Histopathologic Study.

A. R. HOLLENDER, M.D.

(Abstract)

While many of the principal physiologic changes of normal aging are known, others still remain in the realm of the unknown. Our knowledge of the changes resulting in certain body tissues from aging is likewise obscure. In order to throw some light on the subject, as far as the nasal mucous membrances are concerned, this present study was undertaken.

The problem had to be approached cautiously because of the numerous factors involved. Since the nasal mucosa is constantly exposed to various irritants, this fact had to be taken into consideration, for it was realized that nearly every elderly person has experienced several inflammatory attacks on his nasal mucosa during his lifetime. Further, the material for this study was obtained from cadavers of persons whose fatal disease may have contributed to some of the structural changes in the nasal tissues.

Histopathologic study was made of the inferior turbinates removed from 23 cadavers of persons between 50 and 90 years of age. Interrupted serial sections were made of the turbinal specimens and prepared for study with appropriate stains.

All types of epithelium were seen. In general the normal accumulation of lymphatic tissue in the subepithelial layer was decreased and replaced by connective tissue which was more abundant than is usually found in specimens from juvenile individuals. When the connective tissue was firm, there was frequently a marked depo-

sition of hyaline. When bone was present in the specimens, osteogenesis was frequently observed, osteoclasis being only occasionally seen. The corpus cavernosum did not show any conspicuous signs, although there was a definite variety of the quantitative development. From the findings in two cases one is able to conclude that the nasal corpus cavernosum may present a hypertrophy in that its blood spaces become increased in number at the expense of the interstitial and subepithelial tissues of the mucosa. Since in both cases there was a liver cirrhosis, one might think of varicosities in the cavernous tissue of the nose resembling the varicosities often found in the esophagus in liver cirrhosis. The glands were found in various stages of activity. Frequently the excretory ducts of the glands were situated in the depths of the lamina propria of the mucosa. The interalveolar tissue of the glands was increased and hyalinized, resulting in compression and atrophy of the glands.

In individuals who died from hypertensive heart disease, the arterioles of the nasal mucosa presented marked hyaline deposits in the adventitia similar to that in other organs of the body.

From this study one is able to conclude that the following are the important histopathologic findings:

1. Involution of the subepithelial lymphatic tissue.

2. Proliferation and hyalinization of the connective tissue with resulting atrophy of the glands.

3. Hyalinization of the adventitia of the arterioles.

This study opens new avenues for examining the changes in the nasal mucosa incident to, or produced by, aging. It is hoped to present a more complete report at a later time, but meanwhile others should become interested in this problem.

Postthyroidectomy, Bilateral Laryngeal Paralysis Complicated By Myxedema and Hypoparathyroidism.

PAUL H. HOLINGER, M.D.

(Abstract)

Increased interest in the problem of bilateral vocal cord paralysis associated with thyroid surgery may be accredited to the operation devised by King. Proper restoration of function is primarily a surgical problem. However, medical aspects of the problem require

more attention than is generally directed toward them, and in many instances may be responsible for the success or failure of the surgical procedure.

The section of both recurrent laryngeal nerves in itself usually indicates that an extensive operative procedure has been done. Consequently, one may expect to find associated with bilateral paralysis following thyroidectomy varying degrees of myxedema and hypoparathyroidism. This possibility has been shown to occur in each of six patients recently referred for relief of the respiratory obstruction associated with their bilateral paralysis. Medical examination in each showed varying degrees of myxedema as evidenced by the thick skin, low voltage electrocardiogram, high serum cholesterol, anemia, mental sluggishness or depression. The basal metabolic rate determination was considered erroneous because of leaks around the tracheotomy tube if the patient was tracheotomized, and by the chronic anoxemia of those not tracheotomized. Evidence of parathyroid tetany was found in each as evidenced by the history of cramps in the hands and feet, psychoses, and by the laboratory finding of extremely low blood calcium and high phosphorus.

Treatment of the thyroid deficiency consists of administration of thyroid substance; this administration should be controlled by the clinical course and the blood studies of the patient rather than by basal metabolic rate determinations. Treatment of the parathyroid deficiency consists of administration of calcium lactate or gluconate and vitamin D or dihydrotachysterol, the amount given to be controlled by the blood calcium studies. In two of the operative patients who had a lateral transposition of the arytenoid, symptoms of obstruction recurred six months and one year later. They were found to have discontinued the use of thyroid extract and calcium. Improvement followed return of their blood chemistry to normal levels. Because of these findings, it is felt that the association of myxedema and tetany with bilateral recurrent laryngeal nerve paralysis is a fairly frequent one, and that the best surgical results can be obtained only after correction of endocrine deficiencies.

Hereditary Hemorrhagic Telangiectasia

MAX H. CUTLER, M.D.

(Abstract)

The case is reported of a 54-year-old white man with hereditary hemorrhagic telangiectasia, with a history of severe exsanguinating

epistaxis requiring, during the past four years, a total of 54 blood transfusions. Control of bleeding was established by balloon catheter, which allowed the discovery and ligation of a large arterial bleeding point. The patient has been relatively free from epistaxis since this was done.

Clinically, there are three groups of cases differentiated as to degree of symptomatology:

1. Nonbleeding cases in which telangiectasis is in evidence. These are usually discovered in a survey of families in which other members have severe bleeding. These persons usually do not come for aid.

2. Cases of mild to moderate epistaxis, which can be controlled by local measures such as cautery and cotton packs. They are self-limiting. These patients do well with albolene for crusts and radium therapy.

3. Cases of the severe exsanguinating type, in which a traumatic background, surgical or otherwise, may be present. In this group there is a mortality rate of four per cent. This type usually occurs in the age group from 45 to 60, when vascular changes are greater in occurrence. There is erosion by the telangiectasia of large or major arterial twigs. These patients require repeated transfusions, through which intolerance may develop. Treatment with radium at times alleviates the condition, while in other cases radium is of no value.

Carcinoma of the Maxillary Sinus

MAURICE F. SNITMAN, M.D.

(Abstract)

The early treatment of carcinoma of the maxillary sinus was entirely surgical, but results were uniformly bad, since the lesion was well advanced when first seen. With the advent of x-ray and radium therapy, more encouraging results were obtained by a combination of surgery and irradiation. The irradiation was usually in the form of radium implanted in the operated field. Since 1931, protracted fractionated irradiation combined with limited surgery of the antrum for purposes of drainage and inspection, has led to a substantial improvement in results. Del Regato in 1937 reported five to fifteen

year cures in four of ten inoperable cases. Dr. Danely P. Slaughter, director of the Tumor Clinic of the University of Illinois. suggested the supplemental application of intracavitary radium and this procedure was followed on two recent cases.

CASE 1. A. R., a white woman, aged 42, was first seen April 3, 1943, with a complaint of expectoration of bright red blood in the morning for four weeks and swelling of the left malar region for six weeks. There was a history of numbness of the left side of the face in 1937, associated with postnasal discharge and frequent sore throats. Gradually severe headaches set in which persisted for one year; there were frequent upper respiratory infections. All upper teeth were removed in 1942. In February 1943, in conjunction with a severe upper respiratory infection with nasal obstruction, there was expectoration of blood and slight redness of the left eye, with a sensation of "crawling" over the left cheek. For a year prior to first examination she had occasionally noted blood-tinged mucus on expectoration.

On examination there was distinct swelling over the left cheek, below and lateral to the eye; there was some limitation of lateral movement of the left eye. On transillumination, the left antrum was dark; on irrigation there were no significant findings. X-ray examination showed increased density over the left antrum with distinctive change in the malar bone suggestive of tumor formation.

On April 28, 1943, a Caldwell-Luc operation disclosed a mass filling the antrum, a biopsy of which showed cylindrical cell carcinoma. 4800 R were given to two portals over the left antrum and temple between May 24 and June 26. On July 1, 1943, under general anesthesia, the external carotid artery was ligated and the antrum reopened, with removal of the greater part of the canine wall and alveolar process. Several weeks later a small fluctuating swelling about 1.5 cm. in diameter developed over the outer angle of the left orbit. This disappeared following insertion of two radon seeds of 1.5 mc each. On July 30, 1500 mc hours of radium was placed into the antral cavity. There was uneventful healing with a permanent antral fistula. Examination in February 1944 showed a smooth-lined cavity which has shown the same appearance for the past several months.

CASE 2. F. W. H., a 67-year-old man, was first seen in October 1943, complaining of swelling of the right cheek, chiefly in the malar region, with narrowing of the palpebral fissure; in addition, there was a large swelling of the right upper cervical node and a smaller

one in the lower part of the posterior triangle. Fourteen months before, he noted itching, burning and smarting over the right cheek; the swelling was not apparent until about one month prior to consultation in October. He had lost ten pounds in the past few months.

Examination of the nose and mouth gave negative findings. X-ray films showed involvement of the malar bone and floor of the orbit. The antrum was cloudy, and the nodes in the upper cervical region and posterior triangle were seen.

On October 25, 1943, under general intratracheal anesthesia, the right external carotid artery was ligated and the right antrum opened sublabially. The cavity of the antrum was filled with friable degenerated tissue; this was cleaned out and the anterolateral surface of the antrum was removed. Seven radium needles, each 10 mgm, were arranged in a circle in the antrum cavity, and left in place for 28.5 hours, giving a total dose of 2000 mgm hours. Into the upper cervical node 13 radon seeds with a total value of 2600 M.C.H. were implanted directly through the skin. The lower node received 4 radon seeds (800 M.C.H.), and disappeared within 8 days. On November 10, 7 more radon seeds (1350 M.C.H.) were implanted into the upper node, and 10 days later an epidermite developed in this area. From November 3 to December 13, he received daily external radiation to the antrum through two portals until a total dose of 5260 R was given. Another series of daily treatments from December 18 to January 12 totaled 3800 R.

Examination in the latter part of February 1944 disclosed that a fibrotic nodule had replaced the large upper cervical node. The antral fistula permitted free inspection of the cavity, which showed a fair degree of radionecrosis.

The immediate result has been quite satisfactory in each of the two cases. Obviously, more time and a larger series will be necessary for determination of the efficacy of intracavitary radium supplemental to external protracted fractionated irradiation and limited sinus surgery.

Sphenoid Sinus Disease

O. E. VAN ALYEA, M.D.

(This paper appears in full on page 493)

DISCUSSION OF THE PAPERS OF MARCH 6, 1944

DR. HOWARD C. BALLENGER: Dr. Snitman mentioned paresthesia as a prominent symptom in two cases. In one he stated the tumor was in the upper wall of the sinus. I would like to know if the growth originated in the roof of the maxillary sinus in the second case as well. Paresthesia and anesthesia of the side of the face seem to be early symptoms of malignant growths originating in the upper half of the maxillary sinus. This point needs stressing as an important diagnostic symptom.

DR. ALFRED LEWY: I would like to report a case of sphenoid disease in which the diagnosis was missed. This was in a middle-aged woman who had undergone some mental changes. She had a right-sided otitis media of unknown duration, with nystagmus to the left and vomiting. A diagnosis of right suppurative labyrinthitis was made. Operation was refused and she soon developed meningitis and died. Autopsy showed the meninges in the region of the right temporal bone to be perfectly normal. It was practically a unilateral meningitis limited to the left side. The source was found in the necrotic roof of the left sphenoid sinus, which led to the infected sphenoid cavity.

DR. THOMAS C. GALLOWAY: We have had quite a few cases of Osler's disease at County Hospital, but have not been able to do much for them. One was a physician who had been to many clinics but had increasingly severe hemorrhages. We have treated some of the bleeding lesions with desiccating diathermy, but I have not seen much result from radium or radon, and most cases seem to go on a downhill course. One of our residents found one family in which seven or eight members had the disease.

With reference to carcinoma of the antrum, in the presentation of individual cases it is easy to show some beautiful results of a few months' duration. However, we should be interested in five-year cures. There have been at least three important series treated with surgery — usually electrocoagulation — combined with irradiation, with roughly 37 per cent cures, and the combined method would still seem the best.

In those cases which produce external symptoms, my experience has been that destruction of the invaded bone by surgical diathermy is required.

DR. SAMUEL SALINGER: I would like to point out, in connection with carcinoma of the antrum, that the Mayo Clinic report makes a sharp distinction between carcinoma involving the lower half of the antrum and that which takes in the upper half, showing a definitely better prognosis in the former. I agree that judging by the results in reports of these large series of cases, it is evident that the best results have been obtained by surgical diathermy followed by irradiation.

I was impressed by frequent mention of the use of contrast media for diagnosis. I have been a strong believer in this procedure and advocate its use whenever possible, particularly in doubtful cases or where surgery is contemplated.

The sphenoid sinus, because of its remoteness, has too often been neglected. There has been a high incidence of intracranial complications traceable to involvement of the sphenoid which have been frequently overlooked. The report by Pickworth and Graves, in connection with a study of the relationship between sinus disease and mental disorders, mentions the investigation of tissue removed from the sinus. In numerous instances they found attenuated streptococci deep in the mucosa and even in the bony lacunae. I think if we look for sphenoid sinus disease more frequently we will find it.

DR. HANS BRUNNER: The presentation of Dr. Hollender exhibited genuine pathology of the nasal mucosa. Too frequently pathologic changes of the nasal mucosa are described to fit the personal interpretation of the author. The pathologic character of the hyaline deposits which were demonstrated cannot be doubted, and on the basis of these findings this study possesses considerable merit. The practical conclusions which can be drawn from this investigation may eventually prove valuable.

Dr. Holinger's paper was interesting. It proves that in instances of bilateral abductor paralysis following goiter operations, the surgical repair of the nerve function does not solve the entire problem. If his findings are confirmed by further observations, one must assume that the paramedian position of the vocal cords following injuries of the laryngeal recurrent nerve, is not only due to the surgical lesion of the nerve but also to a laryngospasm which, in turn, is due to damage of the parathyroids. The latter part of this concept is supported by many endocrinologists who believe that laryngospasm in children as well as in adults is, as a rule, attributable to a hypo-

function of the parathyroids, even if there are no other symptoms or tetany.

Dr. Cutler, of course, does not consider his case as definitely cured. Osler's disease is a systemic disease which involves the walls of the capillaries and if the hemorrhage is stopped in one place it may occur in another place at any time.

Dr. Snitman mentioned the poor results obtained in the past by surgery of antrum carcinomas. In those days intratracheal anesthesia was not employed. Furthermore, patients were usually operated on by general surgeons who made a complete resection of the maxilla. In many instances the fatality was due to pneumonia. At present the prognosis is better. Nevertheless, it depends much on the structure of the tumor and on the involvement of the ethmoids. So far as the glands are concerned, one must keep in mind that enlargement of the glands is not always due to the malignancy. Frequently the enlargement is caused by inflammation, in which case the glands may contain tubercle-like formations.

DR. M. F. SNITMAN (closing): Both these patients showed involvement of the malar bone and presented almost similar complaints of paresthesia over the face in the form of itching and crawling sensations.

Enlarged cervical nodes may certainly be tuberculous, but in all cases treatment by radon seed implantation is preceded by positive aspiration biopsy findings. Dr. Galloway should be complimented for the statement he made in a recent article in the *Annals of Otology, Rhinology and Laryngology* emphasizing the need for teamwork in the management of malignancy.

These are both very recent cases and are presented as immediate results, where external fractionated protracted irradiation is supplemented by limited exposure of the antrum and intracavitary radium. If we can secure the advantages of protracted irradiation in the management of carcinoma of the antrum, as is the case with malignancy in other regions, we may obtain an increasing rate of cures. It is interesting to note that Watson of the Memorial Hospital reports that every cured case during the past seven years has had roentgenotherapy, and in six instances this was the only treatment employed.

Regarding bone invasion, Willis, in his text "The Spread of Tumors in the Human Body," states that the carcinoma involves the bone after the latter has undergone inflammatory changes as a result of infection.

Meeting of Monday, April 3, 1944

THE PRESIDENT, DR. SAMUEL J. PEARLMAN, IN THE CHAIR

Reduced Atmospheric Pressure in the Treatment of Paranasal Sinusitis.

D. B. BUTLER, M.S., M.D.

G. J. GREENWOOD, D.D.S., M.D.

A. C. IVY, PH.D.

(Abstract)

A series of 125 patients with chronic sinus disease were treated by rapid decompression in a high altitude chamber to an atmospheric pressure of 522.6 mm. Hg. which is equivalent to 10,000 feet above sea level; 2259 treatments were given. Subjective improvement was observed in 89.5 per cent of the patients. A group of 29 patients have been followed by serial x-ray studies after instillation of radiopaque oil. Seventeen patients, or 58.6 per cent, showed improvement in the x-ray picture after decompression treatments. A group of 42 patients were studied by repeated rhinologic examinations. thirty patients, or 71.4 per cent, showed improvement. Decompression therapy is presented as a means of simple nontraumatic treatment of subacute and chronic sinus disease, without making any claims of superiority over other therapeutic procedures, though such may be inferred when the therapeutic benefits most of our patients obtained from other therapies are compared with those of the present procedure.

DISCUSSION

DR. THOMAS GALLOWAY: This study has been made in Dr. Ivy's usual thoroughgoing manner. One question of interest is what effect the lowered pressure at higher altitudes has on blood flow and edema in the sinuses. Would it increase congestion by relieving the pressure on the vessels? If so, that is important. If the decreased density of air pressure is important there should be other methods of obtaining that effect; if this principle applies, there will undoubtedly be some simpler way. For instance, the patient's head could be placed in a negative pressure chamber. Does displacement drainage by posture changes account for any of the beneficial effect?

Dr. Ivy uses the term nontraumatic, apparently in contrast to his idea of office treatment. Yet, suction in the office should not be used to the point of physiologic trauma.

DR. JOHN R. LINDSAY: This has been an interesting presentation. When analyzed, however, the effect of this treatment does not appear to differ from that customarily obtained by other means. Two years ago in our clinic Dr. Judd made a number of observations on the interchange of pressure between the nasal cavity and the maxillary sinus. Normally there is free interchange of pressure at the ostium. In acute and chronic inflammatory conditions there may be a lag in the equalization of pressure, indicating partial blockage at the ostium which, in turn, causes retention of secretions in the sinus.

In such cases the use of a vasoconstrictor in the middle meatus opens the ostium, as indicated by a return of free interchange of pressure. The cilia are able to function again and some mucopus may shortly be expelled into the nose.

It seems that the effect of the altitude changes is similar, in that mucopus that may be lodged partly in the ostium is moved into the meatus, thereby removing the impediment to ciliary action.

Both methods probably serve only as aids to ciliary function. The use of alterations in pressure obviates the possible irritating effect of a vasoconstricting agent.

DR. SAMUEL SALINGER: The striking thing about the charts presented by Dr. Ivy is the difference in percentage of improvement when judged by the objective signs as compared with the subjective symptoms, the improvement being so much less in the former. To my mind this proves what we all know, namely, that any procedure that assists ventilation of the sinuses is bound to be followed by amelioration of the symptoms. When checked by the objective findings, however, we find that the basic condition responsible for the complaint still exists. It is therefore important in evaluating the report that we know the type of case receiving the treatment, since it will be shown that certain types of disease will respond more readily to simple ventilation than others. It may well be that in Dr. Ivy's group the good results were obtained in only one particular type of sinusitis.

My reaction to the modus operandi of Dr. Ivy's experiment is that the reduced air pressure releases secretion clogging the sinus ostia, thus permitting air to enter the cavity and restore normal balance of air pressure, and this in turn facilitates the ciliary activity.

I do not believe infected secretion can be sucked into a sinus unless the pressure is very great, chiefly because the ciliary wave is constantly in the opposite direction. Such a contention would require more definite experimental proof.

DR. O. E. VAN ALYEA: Dr. Ivy has presented a very important series of experiments illustrating problems in physics. I think to apply it clinically we must go a little deeper. We must attempt to analyze some of the causes of chronic sinusitis. It would seem to me that this particular phase of the investigation was not thoroughly carried out. It has been rather well shown that chronic sinusitis does mean poor drainage. I think for proper evaluation it would, of course, be necessary to study the ostia more carefully as well as the adjacent structures. No mention was made as to the condition of the middle meatus in these cases. What was blocking the ostium—thickened mucosa, polyps, edema of allergy, or deviated septum? I think each case should be studied thoroughly before we can evaluate the results he obtained. I do not think you can lump them in a group and establish satisfactory conclusions.

DR. ANDREW C. IVY (closing): I am sorry Dr. Greenwood could not be present. He made the rhinologic examinations and is the one best qualified by experience in our group to answer questions on that subject. As to whether exposure to altitude actually caused drainage by suction, it can be said that such actually occurred in some, but not all, patients during exposure. We have some photographs of the maxillary ostia before and during exposure to altitude which demonstrate that drainage occurred in some instances. As Dr. Salinger points out, the objective findings are fewer and less favorable than the subjective findings of improvement. However, the disappearance of a long-standing headache without a change in objective findings is significant to us, and especially to the patient. We deemed it advisable to ascertain whether clinical subjective improvement could be obtained by the method, before expending time and money on the collection of objective data. In time we shall accumulate more objective data. Of course, we doubt whether any amount of therapy will clear organic changes, it being recalled that our patients had chronic sinus disorders of long standing. However, as Dr. Salinger points out, the therapy may promote drainage by improving ciliary function. As Dr. Galloway suggests, the improvement may be secondary to hyperemia. In fact, we are finding that when patients are exposed to altitude, hyperemia, as indicated by inspection and the actual measurement of the resistance to air flow, occurs, regardless of the fact that patients report subjectively that

nasal breathing is facilitated, which is probably due to decreased air density.

Regarding the passage of infected material into the eustachian tube or sinus, this might occur if the patient was returned to ground level too suddenly, just as it may occur in an aviator in a power dive or rapid descent. This might occur if a patient or subject were placed in a pressure chamber, as was done by Dr. Behnke, and the pressure increased too rapidly.

As suggested, we might obtain better drainage by requesting the patient to place the head in more favorable positions. We have not introduced that factor into the therapeutic procedure. However, gas is in the sinus and in the fluid in the sinus, and its expansion on being exposed to reduced pressure could facilitate drainage during the exposure. However, I suspect that some less obvious factor, such as hyperemia, rather than direct suction drainage operates to produce the improvement observed.

The Local Use of Powdered Sulfathiazole in Acute Nasopharyngitis (Common Cold).

HOWARD C. BALLENGER, M.D.

(Abstract)

The local use of the various sulfonamides in treating acute upper respiratory tract infections has been receiving increased attention during the past year, due largely to the introduction of the soluble sodium sulfathiazole (2.5%) in combination with dl-desoxyephedrine (0.125%). Other combinations and forms of the sulfonamides have been used in the nasal cavities and sinuses, varying from the suspension of the microcrystals (5%) in a 1 per cent aqueous solution of paredrine hydrobromide or other solutions or substances, to the pure microcrystalline or powdered forms of the drug dusted or blown into the nasal cavities or sinuses.

The principal objection to the use of the various solutions is the improbability of their remaining in contact with the nasopharyngeal mucosa long enough to be effective, even though used many times in the course of the day. Another valid objection to the use of solutions of the soluble sodium sulfathiazole is their high alkalinity (pH approximately 10) compared with the pH of the nasal mucosa of 5.5 to 6.5. However, the pH of the 2.5 per cent sodium sulfathiazole

in the 0.125 per cent vasoconstrictor dl-desoxyephedrine hydrochloride has a pH of 8.6 to 9.0, still somewhat more alkaline than the nasal mucosa. The powdered or microcrystalline forms of sulfathiazole do not have the objection of an excessive alkalinity.

Sulfanilamide and sulfadiazine are much more soluble in the nasal secretions than sulfathiazole and as a result their time of contact with the nasopharyngeal mucosa would be less than if sulfathiazole were used. The sulfonamides, it is generally conceded, have no inhibiting effect on the initiating virus of the common cold or influenza. However, the activated secondary pathogenic bacteria, such as streptococci, pneumococci, and staphylococci, which do most of the damage and cause most of the complications, are inhibited in most instances. The sulfonamides are effective, but to a lesser degree, against Hemophilus influenzae and hemolytic staphylococci.

During a period of eight months (July 1943 to February 1944, inclusive) approximately 500 attacks of various stages of acute nasopharyngitis were treated in the following manner. An average of about three treatments was given in each attack, although many of the patients had only one treatment. Some patients had two or three attacks during this period.

The nasal cavities were first sprayed with the minimum amount of a 2 per cent aqueous solution of ephedrine hydrochloride to produce sufficient shrinkage of the turbinates and nasal mucosa for a maximum distribution of the powdered sulfathiazole which followed. The powder was blown into the nasal cavities, nasopharynx and pharynx until the visible mucosa was covered with a dry chalky white film. During the process of blowing the powder into the nostrils the patient was asked to inhale slowly through the nose. The tip of the powder blower was then turned up and the powder blown behind the palate into the nasopharynx; then with the tip turned down a small amount was insufflated into the laryngopharynx. If tonsils were present, as much of the sulfathiazole as feasible was blown into the crypts of the tonsils. The patient was asked to refrain from blowing the nose or clearing the throat for as many hours as possible. If the nose was not blown until the next morning, the powder could be demonstrated in the discharge or seen as patches on the nasal mucosa in most instances. Visible evidence of the powder on the pharynx was of much shorter duration than in the nose, irrespective or whether the patient refrained from clearing the throat or not.

The quantity of powder used at each treatment was difficult to estimate, but it was probably much less than 0.3 gm., as less than one

pound of sulfathiazole was used throughout the eight months' treatment period. Much of the powder did not adhere to the mucosa but was blown on through or exhaled.

A presumed sensitivity to sulfathiazole was encountered in three instances, as all three patients developed a local skin rash around the nose or face; this disappeared within 24 hours. No other discomfort was noted. Any patient with a known or suspected sensitivity to sulfathiazole was not treated with the substance.

An exact evaluation of clinical results is impossible, but when the therapeutic effects upon this series of patients are reviewed and compared with an equal number of patients with acute nasopharyngitis treated by other methods, the conclusion is inescapable that the course of the acute infection was shortened and the number of complications was lessened. Further clinical and laboratory investigations are necessary before a satisfactory evaluation of this method of treatment of acute nasopharyngitis can be made.

The objections to the local use of the sulfonamides to the nasopharyngeal mucous membrane would be the same as for their oral or parenteral use, but possibly to a less degree. The most important objections would be the possibility of producing a sensitivity, as has occurred from their use on the skin. However, no known instance of sensitization has been noted in this series of cases. If sensitization should occur, even in a very small percentage, the value of the procedure would be limited.

DISCUSSION

DR. J. V. CASSADY: I have used sulfathiazole locally in otitis media, and in the antrum after irrigation. I think many patients who use it promiscuously in common colds have more nasal congestion and more irritation afterward than from ephedrine or drugs that do not have sulfathiazole in them.

DR. SAMUEL SALINGER: I appreciate and applaud Dr. Ballenger's report because he drew no conclusions. When we consider the various factors that enter into the tabulation of some 500 cases of sore throat and rhinitis, it is extremely difficult to appraise accurately one therapeutic measure without numerous controls, which in this instance were not employed. We must admit that Dr. Ballenger did note that his cases seemed to get well more quickly than had been his experience previously with other measures, and since he failed to see any deleterious effects from the treatment, one must inevitably be impressed.

DR. HOWARD C. BALLENGER (closing): As I said before, the most valid objection to the use of any sulfonamide on the nasopharyngeal mucous membrane would be the possibility of creating a sensitization. However, no known case of sensitization has been observed in this group.

Xanthomatosis—Lipoid Granulomatosis of the Temporal Bone.

M. A. GLATT, M.D.

(Abstract)

A man, aged 36, complained of severe left-sided temporal pain, dizziness, deafness and extreme weakness, with no fever, glandular enlargement or discharge from the ear. X-ray examination showed an area of rarefaction at the temporomandibular joint and the squama, but not involving the mastoid cells.

Observation for a period of three months led to the conclusion that the lesion was either a malignancy or xanthoma. At operation a greenish-brown sequestrum was removed which exposed the brain substance. Recovery was uneventful. Histologic studies proved it to be a lipoid granulomatous xanthoma.

The pathogenesis in this lesion is due to either disturbed fat metabolism which produces an infiltration of cholesterol in the reticulo-endothelial cells, or to disturbed equilibrium of the fat content within the endothelial cells. The characteristic histologic feature is the presence of "xanthoma or foam cells." This type of granuloma may invade any portion of the skull, including the paranasal sinuses, in solitary or multiple areas. In the temporal bone it may involve the squama, petrosa and the dura before it progresses to the mastoid air cells.

The symptoms are not typical of ordinary surgical mastoiditis nor of malignancy; nevertheless, clinically the differentiation is not easily made. In the presence of systemic disturbances, known as Hand-Christian-Schüller's syndrome, such as exophthalmus, diabetes insipidus, or the finding of other bone lesions in the skull which show an irregular outline with sharp borders on the x-ray film, the diagnosis of xanthoma should not be difficult. In a solitary lesion with indefinite borders, diagnosis can be made only on finding of xanthoma cells in the histologic studies.

It is important to keep in mind the possibility of this lesion in operative cases which do not heal well and in which other factors have been ruled out. Surgical procedures should not be undertaken. X-ray therapy has a favorable influence in producing a fibrosis of the xanthoma lesions.

DISCUSSION

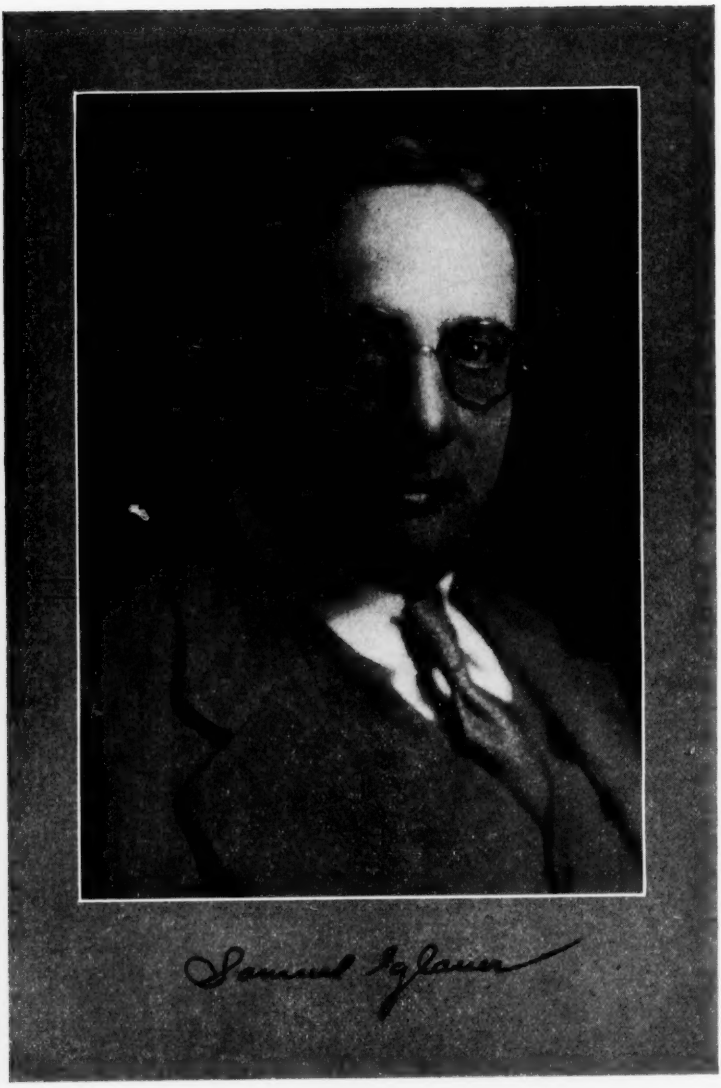
DR. JOHN R. LINDSAY: I treated a case similar to Dr. Glatt's a few years ago. A young man presented himself with a chronic discharge from one ear of several months' duration, with circumscribed labyrinthitis and a very active fistula response. He had in addition a small cyst in the occipital bone on the same side and another in the scapula. Both lungs showed evidence of fibrosis.

A modified radical operation on the mastoid was followed by complete healing with an intact drum, and preservation of function. The labyrinthine fistula healed over. The cysts in the occipital bone and the scapula were also cleaned out. The final diagnosis was xanthomatosis. Up until a year or so ago the patient had developed no further difficulties.

DR. THOMAS C. GALLOWAY: I should like to compliment Dr. Glatt on a fine presentation of a difficult subject. I think this disease has been overlooked frequently. We have seen several cases at County Hospital in recent years, in one of which the involvement was in the frontal bone involving the sinus, in an infant.

DR. M. A. GLATT (closing): My impression is that in this case the lesion may have started from the dura. There were no other bone lesions. Xanthoma may involve the bone marrow and produce bone changes, but it is not primarily a disease of the blood-forming organs. Most of these patients do not show any change in the blood picture.

This is a rare disease, but undoubtedly there are cases on which operation has been performed and in which a histologic diagnosis is not made because of spontaneous healing. I feel that histologic study of the removed tissue should be more carefully done. Usually we take cultures, but tissues are rarely sectioned for study. If this were done we might find more cases. I would hestitate to apply preoperative aspiration to the skull where the dura might be involved, as a diagnostic measure. I believe surgical biopsy to be preferable.


Samuel Sylvan

SAMUEL IGLAUER, M.D.

1871-1944

Samuel Iglauer died at The Jewish Hospital in Cincinnati on June 23, 1944, at the age of 73 years, following a cerebral hemorrhage five days previous. He was born in Cincinnati, the son of Arnold Iglauer of Bavaria, Germany, and Delia Fechheimer of Richmond, Kentucky.

He was educated at the local Cincinnati school and received a Bachelor of Science degree from the University of Cincinnati in 1895. He was graduated in medicine from the Medical College of Ohio in 1898.

Dr. Iglauer's entire medical life was spent in Cincinnati where he held the Professorship of Otolaryngology, College of Medicine, University of Cincinnati, from 1930 until his death, and was Director of Otolaryngology at the Cincinnati General Hospital and Children's Hospital during the same period. He was also Director of Otolaryngology at the Jewish Hospital.

Membership in national medical societies included that of the American Medical Association, serving as Chairman of the Section of Otology, Rhinology and Laryngology (1925-1926); the American Laryngological Association; the American Laryngological, Rhinological and Otological Society, serving as vice president of the Middle Section in 1920, and as member of the Council (1936-1938); the American Academy of Ophthalmology and Otolaryngology, serving as vice president in 1935; the American Broncho-Esophagological Association, serving as president (1923-1924); the American College of Surgeons; the Ohio State Medical Association; the Academy of Medicine of Cincinnati, serving as president in 1933-1934; the Cincinnati Otolaryngologic Society, serving as president (1939-1940); the Cincinnati Anti-Tuberculosis League of which he was a director; the American College of Chest Physicians; the American Board of Plastic Surgery; and American Association for the Advancement of Science.

Nonmedical activities included membership in the Cincinnati University Club; the Cincinnati Country Club; the Losantiville Club; the Cincinnati Torch Club; the Cincinnati Lodge Number 4, B'Nai B'rith; and the University of Cincinnati Chapter of Sigma Xi.

He served in the U. S. Army Service Medical Corps from 1918 to 1919.

Dr. Iglauer was a pioneer in the use of the bronchoscope and was one of the first to note the importance of roentgenographs in the study of mastoiditis. He held a prominent place in the field of plastic surgery and published some of the first articles on the use of lipiodol in the study of diseases of the lungs. He was an authority on the anatomy of the spaces of the neck and published many valuable papers on this subject.

He was very fond of athletics and was always a cheerful companion at golf. He had a great sense of humor and never spoke unkindly to his associates, no matter what the situation. He enjoyed going to the various medical meetings, not only to take part in scientific discussions but to enjoy his colleagues and friends of which he had many.

He is survived by his wife, and two daughters, Dr. Helen I. Glueck and Mrs. Josephine I. Joseph, both of whom live in Cincinnati.

HENRY M. GOODYEAR.

PUBLICATIONS OF DR. SAMUEL IGLAUER

1. Clinical Value of Radiography of Mastoid Region, J. A. M. A., Sept. 1909.

2. Use of Electro-Magnets in the Extraction of Metallic Bodies from the Trachea and Bronchi with Report of Cases, Laryngoscope, Jan. 1914.

3. Value of Roentgenography in Diagnosis of Diseases of the Larynx and Trachea, J. A. M. A., Dec. 1916.

4. The Oblique Method of Roentgenography of the Ethmoid and Sphenoid Cells, J. A. M. A., Dec. 1916.

5. Some Original Methods of Treatment of Laryngeal Stenosis, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Dec. 1918.

6. The Report of a Case of Laryngocele, New York M. J., Mar. 1921.

7. Epiglottidectomy for Relief of Congenital Laryngeal Stridor, Laryngoscope, Jan. 1922.

8. Perforation of the Esophagus by a Foreign Body with Report of a Case Presenting Unusual X-ray Signs, Laryngoscope, Oct. 1924.

9. Use of Injected Iodized Oil in Roentgen-Ray Diagnosis, J. A. M. A., June 1926.

10. X-Ray Injury to the Larynx—Report of Case, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Mar. 1927.

11. Use of Iodized Oil in Diagnosis of Pulmonary Lesions, Surg. Gyn. & Obs., July 1927.

12. Advantages of Brominized Oil in Bronchography in Tuberculous Patients, J. A. M. A., April 1928.

13. Pneumocele of the Frontal Sinus, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Mar. 1930.

14. Deep Suppuration in the Pharynx and Neck as it Concerns the Laryngologist, J. Michigan State Med. Soc., June 1931.

15. Impermeable Stricture of the Esophagus Relieved by Retrograde Bouginage with the Aid of a Cystocope Inserted Through a Gastrostomy, ANNALS OF OTOLOGY, RHINOLOGY AND LAGYNGOLOGY, Dec. 1932.

16. Objective Tinnitus Aurium, Arch. Otolaryng., Aug. 1933.

17. Retropharyngeal Abscess, Cyclopedia of Med., Philadelphia, F. A. Davis Co., Vol. IX, 1934; Vol. XIII, 1936.

18. Deep Infections in the Neck and Mediastinum, Kansas City S. W. Clin. Soc. Bull., Apr. 1935.

19. Surgical Approaches to Deep Suppuration in the Neck and Posterior Mediastinum, Arch. Otolaryng., June 1935.

20. A New Procedure for the Treatment of Web in the Larynx—Report of a Case, Arch. Otolaryng., 22:597-602 (Nov.) 1935.

21. Heart-Block Periodically Induced by the Swallowing of Food in a Patient with Cardiospasm (Vagovagal Syncope), ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 45:875 (Sept.) 1936.

22. Acute Suppuration in the Deep Structures of the Neck, Practitioner's Series of Medicine and Surgery, Vol. XI, 1937.

23. Pulmonary Collapse Following Tonsillectomy under Local Anesthesia, Arch. Otolaryng. 25:382-388 (April) 1937.

24. A Case of Dysphagia Due to an Osteochondroma of the Cervical Spine—Osteotomy—Recovery, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 47: 799 (Sept.) 1938.

25. Rupture of the Esophagus with Spontaneous Recovery of the Patient, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 47:1083-88 (Dec.) 1938.

26. Severe Injury to the Larynx Resulting from the Indwelling Duodenal Tube (Case Reports), Dr. William F. Molt, Indianapolis, Ind., co-author, Trans. Amer. Bronchoscopic Society, 1939.

27. Anatomic-Pathologic Studies of Retropharyngeal Abscess, Arch. Otolaryng. 33:31-34 (Jan.) 1941.

28. Indications for Surgery in Nasal Sinus Disease, Diseases of the Eye, Ear, Nose and Throat, Vol. 1, No. 5, May 1941.

29. Recent Literature on Retropharyngeal Abscess, Cyclopedia of Medicine, Philadelphia, F. A. Davis Co.

30. Relation of the Respiratory Passages to Anesthesia, Anesthesiology.

31. Deep Infections in the Neck, Ohio State M. J.

32. The Use of Preserved Human Cartilage in Reconstructive Facial Surgery, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 50:1072 (Dec.) 1941.

33. Roentgenology of the Neck and Mediastinum, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 50:790 (Sept.) 1941.

34. Septic Thrombophlebitis of the Neck, Arch. Otolaryng. 36:381, 1942.

35. Two Cases of Osteomyelitis Treated with Penicillin, Laryngoscope.

36. Bronchoscopy As A Diagnostic and Therapeutic Procedure, Nebraska State M. J. 28:340-344 (Nov.) 1943.

And numerous other papers.

Books Received

Tuberculosis of the Ear, Nose and Throat: Including the Larynx, the Trachea, and the Bronchi.

By Mervin C. Myerson, M.D., New York City. Pp. ix+291, illustrated. Springfield, Illinois, Charles C. Thomas, 1944. (Price $5.50)

This thorough treatise is based upon a careful study of 1,000 cases of laryngeal tuberculosis seen by the author, occurring in more than 10,000 cases of pulmonary tuberculosis, an incidence of approximately ten per cent.

His evaluation of symptoms, his histologic studies and painstaking diagnostic procedures ably substantiate his well ordered conclusions which direct the treatment of these conditions. He is neither overly sanguine, nor depressingly conservative in his attitude. He stresses the universally accepted axiom that complete bodily and vocal rest are the most valuable assets which we have at our disposal for the treatment of this condition. He might have laid more stress on the equally valuable adjunct of the proper psychological treatment of these patients in order to support their morale. Such psychological assets as heliotherapy, chaulmoogra oil and mildly stimulating oils used locally, while of questionable value therapeutically, are really very important in the minds of individuals who must devote many months, or possibly years, to the accomplishment of a cure.

The subject is well handled throughout, not only in his description of lesions of the larynx, but also in those of the pharynx, the nose, the middle ear, the esophagus and the tracheobronchial tree. While the author states that he is elucidating his own views, he quotes freely from many other eminent authorities. His diagrams and photographs illustrate well the conditions which he describes. The chapters and text are well arranged and the subject matter very accessible. The author is to be congratulated on a fine piece of work for which there is a definite need.

McMahon.

Minor Surgery.

Edited by Humphrey Rolleston and Alan Moncrieff. Pp. viii+174, illustrated. New York, The Philosophical Library, 1944. (Price $5.00)

In the two short chapters devoted to surgical procedures of the ear, nose and throat, the authors strike a safe note in their admonition that "operations on the nose and throat, however small, should not be undertaken lightly and without due consideration", which good advice is later stressed in their discussion on tonsillectomy when they state that "the tonsil and adenoid operation is one of the most difficult to do well and skilfully".

They devote a few carefully worded paragraphs to anesthesia, fracture of the nasal bones, nasal hemorrhage, handling of foreign bodies in the nose, peritonsillar abscess and tonsillectomy. Under each paragraph they describe one or two methods which are orthodox and generally acceptable.

In their consideration of conditions involving the external and middle ear their advice is equally sound, and commendably conservative. They mention the removal of cerumen, discharge and debris from the external auditory canal, furunculosis, the hazards of politzerization and the safety and accuracy of catheterization of the eustachian tube, and the indications for and technique of safe myringotomy. They have succeeded in compressing an extraordinary amount of sound advice in a small, well-arranged volume.

McMAHON.

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MEMORY

OF

LEE WALLACE DEAN, SR.
SC.B., SC.M., M.D.

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